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Growth Through Agricultural Progress

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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CONTENTS

1. Chemical control of weather fleck in flue-cured tobacco
E. K. WALKER 583

2. Some hosts of Erwinia carnegieana
STANLEY M. ALCORN 587

3. Low temperature injury as a contributing factor in Cytospora invasion of plum trees
A. W. HELTON 591

4. Conditions favorable for adsorption and elution of tobacco mosaic virus in an ECTEOLA-cellulose column
T. E. RAWLINS 598

5. Yellow mottle decline of coconuts in the Territory of Guam
OTTO A. REINKING 599

6. Response of Zinnia varieties to tobacco ringspot virus
J. G. KARAS, et al. 605

7. The rapid determination of necrotic ring spot and sour cherry yellows viruses of stone fruits
R. M. GILMER 608

8. The frequency of necrotic ring spot, sour cherry yellows, and green ring mottle viruses in naturally infected sweet and sour cherry orchard trees
R. M. GILMER 612

9. Pythium ultimum and Helminthosporium vagans as foliar pathogens of Gramineae
LAURENCE D. MOORE and HOUSTON B. COUCH 616

10. Performance of certain nematocides and nematocide-fertilizer mixtures applied to vegetable crops in North Carolina
ROBERT AYCOCK and J. N. SASSER 620

11. Irish potato seed piece treatment with various chemicals
J. B. SINCLAIR 625

12. Comparison of standard fungicides recommended for control of powdery mildew of roses
IRA W. DEEP and ALLAN BARTLETT. 628

13. Wheat rust during the Spanish era in California
RALPH E. SMITH and JACOB N. BOWMAN 632

14. Boron deficiency evident in Oregon holly orchards
A. N. ROBERTS, et al. 634

15. 2,6-dichloro-4-nitroaniline effective against Rhizopus fruit rot of sweet cherries
JOSEPH M. OGAWA, et al. 636

16. Infection of artificially inoculated shortleaf pine hybrids with fusiform rust
F. F. JEWELL. 639

17. Resistance to powdery mildew in leafroll-affected grapevines
A. C. GOHEEN and W. C. SCHNATHORST 641

18. The pathogenicity of certain species of Helminthosporium to species of the Gramineae
R. R. NELSON and D. M. KLINE 644

19. Yellow bud virus endemic along California coast
N. W. FRAZIER, et al. 649

20. Influences of surfactants and the miticide Aramite on control of rose blackspot by Phaltan
JOHN G. PALMER and T. J. HENNEBERRY 652

21. Reaction of wheat seedlings to new isolates of wheat stem rust
JAMES D. MILLER and DONALD M. STEWART 657

22. Susceptibility of detached Royal apricot fruits to various plant pathogens
THOMAS H. STANTON and JOSEPH M. OGAWA 659

23. Fomes annosus causes severe damage to thinned slash pine plantation in south Georgia
CHARLES H. DRIVER and T. R. DELL 660

24. Distribution and prevalence of physiologic races of leaf rust of wheat, Puccinia recondita Rob. ex Desm., in the United States in 1960
C. O. JOHNSTON 661

25. Studies of the effect of straw cover and method of inoculation on dwarf bunt of winter wheat in Utah
WADE G. DEWEY. 663

26. Occurrence of Cercospora bougainvilleae in El Salvador
OSCAR ANCALMO 666

27. Book Review (Monograph of Monochaetia and Pestalotia)
JOHN A. STEVENSON 666

CHEMICAL CONTROL OF WEATHER FLECK IN FLUE-CURED TOBACCO[1]

E. K. Walker[2,3]

Summary

In 1959 and 1960 a number of compounds and commercially formulated fungicides, each chosen as a potential antioxidant, were sprayed on flue-cured tobacco in an attempt to prevent weather fleck in the field, and ozone damage in a fumigation chamber. Fifty percent dichlone wettable powder (Phygon XL) at 2 pounds/100 gallons of water, applied to both leaf surfaces, gave highly effective control in both field and chamber studies. Applications to the upper surface only were somewhat less effective. Certain of the other materials, although less effective than Phygon XL, gave indications of at least partially preventing both weather fleck and ozone damage.

INTRODUCTION

A leaf disorder, commonly known as weather fleck and believed to be physiological in origin, has been a serious problem of flue-cured tobacco in Ontario since 1954. The disorder appears to be similar to the weather fleck of cigar-wrapper tobacco described by Heggestad and Middleton (1), and the oxidant stipple of grape described by Richards, et al. (2). The small irregularly-shaped spots are initially black, but later turn brown and greyish-white. Injury is first apparent in the palisade mesophyll, but within 12 hours the upper epidermis breaks down and the spots show through on the surface. Severe injury usually occurs only on leaves at, or close to, physiological maturity under stable weather conditions.

Incidence of the disorders in Connecticut (1) and in California (2) have been correlated with high concentrations of ozone. The possibility of such a relationship in Ontario is presently under investigation. Concurrently, preventive measures against oxidant damage are being tested. The results of preliminary investigations with antioxidant spray materials for the prevention both of naturally-occurring weather fleck and of ozone damage are reported here.

MATERIALS AND METHODS

In a field trial in 1959, selected materials (Table 1) were sprayed at periodic intervals during the season on the upper leaf surface of eight plants per replication in each of three treatment blocks. In two field trials in 1960, each comprising quadruplicate treatment blocks containing four plant for each replication, materials were used that had been particularly effective in preventing weather fleck in the field in 1959 and ozone damage in a fumigation chamber in 1960. The first of four spray applications to field plots in both years was on July 7, subsequent applications being made at intervals of 10 days. Both leaf surfaces were sprayed in the 1960 trials. All plants used in each year were of the White Gold variety.

Plants to be exposed to ozone were grown in 2-gallon glazed pots to a height of approximately 30 inches. Excessive amounts of water were applied prior to this stage to hasten leaf maturation, since previous studies (5) showed that the incidence of weather fleck increased when the growth period was successively shortened by increasing amounts of irrigation water. Preliminary work indicated that stomatal opening decreased with reduction of soil moisture content, and, since it has been found that the amount of ozone damage is in direct proportion to the extent of stomatal opening (3, 4), the soil in the pots was raised to the field capacity the day prior to fumigation, in order to promote the opening of the stomata.

The temperature and the relative humidity in the fumigation chamber, which had a capacity of 106 cubic feet, were maintained at 75° F and 90%, respectively, during ozone exposure periods by means of an Aminco-Aire unit. Artificial lights in the chamber supplied about 2000 foot candles. Since very little damage had resulted from exposure of plants to ozone in the dark, illumination was considered necessary to stimulate stomatal opening and thereby facilitate entry of ozone into the leaves. In 1960 Taylor, et al. (3) had reached the same conclusion

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[1]Contribution No. 26, Research Station, Research Branch, Canada Department of Agriculture, Harrow, Ontario.

[2]Agronomist, Tobacco Substation, Delhi, Ontario.

[3]Appreciation is expressed to Dr. V. C. Runeckles, Imperial Tobacco Company, Montreal, Quebec, for assistance in selecting spray materials used in this investigation.

Table 1. Spray materials used for control of weather fleck and ozone damage on flue-cured tobacco.

Material	Active ingredient	Manufacturer or formulator
Phygon XL	50% dichlone	Naugatuck Chemicals, Elmira, Ontario.
Karbam Black	76% ferbam	Green Cross Products, Ltd., Montreal, Quebec.
Arasan	50% thiram	E. I. DuPont DeNemours & Co., Wilmington, Delaware.
2-mercaptobenzothiazole	100% 2-mercaptobenzothiazole	Eastman Organic Chemicals Co., Rochester, N. Y.
Dithane M-22	70% maneb	Rohm & Haas Co. Ltd., West Hill, Ontario.
Cragfruit fungicide 341	34% glyodin	Carbide Chemicals Co., Montreal, Quebec.
GC-2466	50% mucochloric anhydride	Allied Chemical Corp., New York, N. Y.
Kojic acid	100% kojic acid	Brickman & Co., Montreal, Quebec.
Tannic acid	100% tannic acid	Fisher Scientific Co., Toronto, Ontario.
Captan 50-W	50% captan	Stauffer Chemical Co., New York, N. Y.
Dithane D-14	19% nabam	Rohm & Haas Co. Ltd., West Hill, Ontario.
Dithane Z-78	65% zineb	Rohm & Haas Co. Ltd., West Hill, Ontario.

Table 2. Effectiveness of spray materials for control of weather fleck in flue-cured tobacco.

Year	Treatment[a]		Index of weather fleck[b]
	Material	Rate/100 gallons of spray	
1959	Phygon XL	3.2 lb.	5.4
	Karbam Black	1.25 lb.	8.9
	Arasan	do.	10.2
	2-mercaptobenzothiazole	do.	11.2
	Dithane M-22	do.	11.6
	Cragfruit fungicide 341	3.0 pt.	11.6
	GC-2466	1.25 lb.	11.7
	Kojic acid	do.	12.0
	Tannic acid	do.	13.7
	Captan 50-W	do.	14.5
	Dithane D-14	4.0 pt.	14.6
	Dithane Z-78	1.25 lb.	15.7
	Control	--	17.2
		L.S.D. .05	8.3
		C.V.	40%
1960	Phygon XL	2.0 lb.	0.4
	Dithane M-22	do.	5.8
	Karbam Black	do.	8.5
	Arasan	do.	8.9
	2-mercaptobenzothiazole	do.	11.2
	Control	-	16.6
		L.S.D. .05	3.9
		C.V.	26%
	Phygon XL	2.0 lb.	0.7
	do.	1.0 lb.	4.0
	do.	0.5 lb.	10.3
	do.	0.25 lb.	14.1
	do.	0.125 lb.	17.9
	Control		23.0
		L.S.D. .05	4.6
		C.V.	26%

[a]Repeated four times in each year.
[b]Final cumulative rating of the season.

Table 3. Effectiveness of spray materials for control of ozone damage in flue-cured tobacco during 1960.

Material	Treatment: Pounds/100 gallons of spray	Coverage[a]	Index of ozone damage
Phygon XL	2.0	P	9.6
do.	do.	C	0.0
Karbam Black	do.	P	11.8
do.	do.	C	19.4
Dithane Z-78	do.	P	24.2
do.	do.	C	10.1
Control	--	--	24.8
Phygon XL	2.0	C	4.2
2-mercaptobenzothiazole	do.	do.	11.2
Dithane M-22	do.	do.	15.6
Kojic acid	do.	do.	14.8
Arasan	do.	do.	25.8
Control	--	--	20.7
Phygon XL	2.8	C	0.7
do.	1.4	do.	0.8
do.	0.7	do.	13.3
do.	0.35	do.	0.4
Control	--	--	19.1

[a]P = partial coverage, upper leaf surface; C = complete coverage, both surfaces.

about the necessity of illumination.

Ozone was generated by pumping air through an electrical discharge developed by a 10,000 volt-transformer. Ozonized air was passed into the chamber through tygon tubing at the rate of 0.5 liter/minute. Measurements of ozone concentration by means of a Mast ozone meter, model 724-1, approximated 20 p.p.h.m., although values varied between 15 and 25 p.p.h.m.

The ozone exposure tests comprised comparisons of the effectiveness of upper versus both leaf surface coverage, different materials, and rates of dichlone (Phygon XL) for prevention of ozone damage. Four plants for each treatment were sprayed immediately prior to being placed in the chamber. Treated plants were exposed to ozone for 12 hours, since the effect of ozone tended to increase with length of exposure up to a maximum at this time.

The incidence of weather fleck in the field and ozone damage in the chamber were measured quantitatively with respect to the number and distribution of spots on affected leaves. A leaf was given the maximum damage rating of 100 if the spots were distributed over the entire leaf surface in sufficient intensity to render the leaf commercially valueless. Approximately 25% of the area of such a leaf would be accounted for as spots. Proportionate ratings were determined for leaves on which the spots were either more discrete or distributed over less than 100% of the leaf surface. Indices of damage, which were calculated for individual plants and for groups of plants by replication or treatment, represent the average concentrations on individual leaves. They were calculated according to the formula

$$I = \frac{(r_1/n_1 + r_2/n_2 + \text{-----} r_x/n_x)}{x}, \text{ where}$$

r = sum of damage ratings on individual leaves and
n = number of leaves rated, in each of x plants.

RESULTS

Several of the spray materials appeared to be effective for control of weather fleck (Table 2). In 1959 the dichlone and ferbam (Karbam Black) treatments appeared to reduce the incidence of weather fleck below that of the control treatment, although the results were too variable to warrant definite conclusions. Nevertheless, that test indicated that some of the materials might provide partial control. The data were considerably less variable in 1960 when sprays were applied to both leaf surfaces. Dichlone was more effective than the other materials in preventing weather fleck, and all materials reduced the incidence of the disorder below that of

the control. With regard to rates of dichlone, weather fleck appeared to decrease proportionately as the rate of application of that material was increased.

Ozone damage produced in the fumigation chamber appeared to be closely similar in appearance to weather fleck as it occurs in nature. The results of ozone exposure tests (Table 3) indicate that some of the materials used may effectively protect plants from ozone damage. Complete spray coverage was more effective than partial coverage only for dichlone and zineb (Dithane Z-78). Conversely, complete coverage was less effective than partial coverage for ferbam. Dichlone appeared to be the most effective of the tested materials for prevention of ozone damage, and except for the relatively high rating for the 0.7-pound rate, there was a consistently minor degree of injury for each rate of dichlone from 0.35 to 2.8 pounds.

DISCUSSION

An apparent similarity in the relative effectiveness of certain spray materials in preventing naturally-occurring weather fleck and artificially-induced injury by ozone, in addition to a similarity in appearance between the natural and induced disorders, indicates that ozone may be one of the contributing causes of weather fleck as it occurs in nature.

A reduction in the amount of the disorder to the extent achieved in 1959, when the spray coverage was incomplete, may represent the degree of control attainable under commercial conditions. Since even a 25% reduction in the incidence of weather fleck undoubtedly would provide sufficient increase in leaf quality to balance the cost of a spraying program, several of the materials could furnish a satisfactory degree of control.

Although complete was preferable to incomplete spray coverage for control of the disorder, it may be objectionable because of spray residues on the lower leaf surfaces. Such residues may adversely affect the physical and chemical properties of the cured leaves. It is unlikely that a significant amount of spray residue could persist on the upper leaf surface for a protracted period, and even that on the lower leaf surface may be broken down at the relatively high curing temperatures. Lack of persistence of residual spray material may be the reason for somewhat poorer control for incomplete as compared with complete spray coverage.

Before any such antioxidant materials can be recommended for commercial use, further investigation is required to compare the degree of control achieved and the effects on leaves from sprays applied at different times and in variable amounts during the season.

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CANADA DEPARTMENT OF AGRICULTURE, HARROW, ONTARIO

SOME HOSTS OF ERWINIA CARNEGIEANA[1]

Stanley M. Alcorn[2]

Summary

Erwinia carnegieana, which causes the bacterial necrosis of saguaro, has been considered restricted to this species. Laboratory tests, however, show that it is pathogenic to the vegetative portions of a number of cacti associated with the saguaro, including prickly-pears, chollas, barrels, and organ-pipes. The organism may also cause disease in plants not native to the United States, including the cacti Pachycereus pringlei and Trichocereus spachianus and the succulents Euphorbia valida and E. obescens. It can effect complete collapse of tomato and squash fruits, and honey-dew melons. Bacilli have been recovered from naturally infected prickly-pear and cholla and from soil in the saguaro forests. These are culturally similar to E. carnegieana and infectious in saguaros.

INTRODUCTION

Bacterial necrosis is prevalent in the stands of saguaro (Carnegiea gigantea (Engelm.) Britt. & Rose) in Arizona. Lightle, et al. (4) could not relate the disease or its causal agent (Erwinia carnegieana Standring) to any other known bacterial diseases of cacti. Species of Opuntia, a number of which are associated with the saguaro (5), did not develop disease following inoculation with the organism. Since these earlier workers (4) did not specify the species in which inoculations were attempted, it seemed worthwhile to inoculate positively identified specimens of Opuntia, and other possible suscepts.

PROCEDURES

Plants and fruits tested (Table 1) were chosen primarily because they represented either morphological types found throughout the saguaro range, or tissues that might be analogous in succulence to the cortical regions of the saguaro. The prickly-pear species, Opuntia engelmannii Salm-Dyck and O. phaeacantha Engelm., were propagated from "pads" while the cylindrical species (chollas) of Opuntia, O. arbuscula Engelm., O. fulgida Engelm., and O. versicolor Engelm., were propagated from "joints." Specimens of Echinocereus engelmannii (Parry) Rümpler and Mammillaria microcarpa Engelm. were transplanted intact from the field. Barrel cacti (Ferocactus wislizenii (Engelm.) Britt. & Rose) were generally field transplants[3]; however, a number of greenhouse propagated seedlings were also tested.

Cephalocereus sartorianus Rose, C. mezcalaensis H. Bravo, Pachycereus pecten-aboriginum (Engelm.) Britt. & Rose, P. pringlei (S. Watson) Britt. & Rose, P. grandis Rose, Trichocereus spachianus (Lemaire) Riccobono[4], Lemaireocereus thurberi (Engelm.) Britt. & Rose (organ-pipe) and saguaro cacti were raised from seed, as were Euphorbia obesa Hook. f. and E. valida N. E. Brown[5]. Seedling plants ranged in height from 1 and 2 inches for barrels and saguaros to 12 inches for Trichocereus. Most were 3 to 4 inches in height. Barrel transplants were 6 to 12 inches high; other transplants or vegetatively propagated cacti inoculated were representative of field-size plants or plant segments. Fruits were obtained from local grocery stores. Inoculum was grown for 18 to 24 hours at 28° to 32° C on potato-dextrose-peptone (plus .1% $CaCO_3$) agar slants. Sterile syringes were used for injecting 1/4 to 1/2 ml of heavy, sterile, distilled-water suspensions of each isolate into each of the plants to be tested. An attempt was made to inject the same volume of inoculum of each of the various

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[1]Arizona Agricultural Experiment Station Technical Paper No. 646.

[2]Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Tucson, Arizona.

Appreciation is given to John A. Booth for his technical assistance.

[3]Appreciation is expressed to the Papago Tribal Council, Sells, Arizona, to W. T. Mendenhall, Arizona Commission of Agriculture and Horticulture, and to the various Superintendents, since 1955, of Saguaro National Monument, Tucson, Arizona, for permission to collect cacti on tribal, state, and National Monument property, respectively.

[4]These seedling cacti were graciously made available by Gates Cactus, Inc., Corona, California.

[5]Euphorbia specimens were donated by George Olin, Tucson, Arizona.

isolates tested in a given species, but the internal pressures that developed upon inoculation would occasionally cause some back-leakage of the bacterial suspension through the injection hole.

Before inoculation all plants were surface sterilized. Usually two or more plants or fruits of each available species were inoculated per isolate per test. Where possible the individual isolates were inoculated into separately potted segments of given plants of the Opuntia species. Otherwise, inoculated segments were not contiguous on the same plant. Appropriate plants, plant-segments, or fruits were injected with comparable volumes of sterile, distilled-water as checks. All inoculations were made under laboratory conditions over a 5-year period. Usually three or four isolates of E. carnegieana were used in each test.

Following inoculation of most species of cacti and squash fruits a limited area of tissue would frequently assume a water-soaked appearance. During subsequent disease evaluations, only those plants showing rot beyond the margins of water-soaking were considered susceptible, even though infectious bacilli might have been recovered from the restricted inoculation site.

Pure cultures of bacilli isolated from typical lesions were inoculated into saguaro seedlings to determine their pathogenicity in that plant.

RESULTS

The earliest symptom in most susceptible plants (Table 1) was the enlargment of the water-soaked area or the development of such an area. This was associated with or closely followed by tissue softening.

Saguaro, organ-pipe, and barrel cacti, tomato and squash fruits, honey-dew melons, and Euphorbia sp. frequently completely collapsed from a malodorous soft rot within 2 to 5 days after inoculation. Occasionally, however, only a portion of a squash fruit would collapse. Similarly, the formation of callus tissue would infrequently limit the rot in individual saguaro and organ-pipe cacti. The elongate Trichocereus spachianus always showed typical and frequently extensive soft rot. With one exception, however, these were eventually contained by callus tissue formation.

Occasionally plants of Cephalocereus sartorianus and Pachycereus pringlei would rot completely. Usually, however, tissue destruction beyond the inoculation site involved only 1/3 to 1/2 of the seedling.

Cephalocereus mezcalaensis was only slightly susceptible. Rot consistently progressed only a few centimeters beyond the inoculation site before "drying-up." Of 17 inoculated plants of Pachycereus pecten-aboriginum, only 1 showed symptoms. Specimens of P. grandis and Mammillaria microcarpa were not susceptible.

In the prickly-pears enlarging water-soaked areas were often bounded by chlorotic margins. Occasionally the epidermis would rupture, the pad would fold over, and a viscous exudate would leak at the site of tissue collapse. More frequently, as the rot spread, the affected portions would dry out, beginning at the initial inoculation site. In the laboratory the bacterial rot would infrequently become systemic. Usually, symptoms were restricted to the inoculated pads, which sometimes dehisced.

Affected cholla segments progressively became discolored, but the tissues generally remained firm and were not observed to "leak." Diseased portions usually soon began to shrivel. In contrast to segments injected with sterile, distilled water, most infected cholla segments dehisced, frequently while the symptoms were still restricted to the inoculation site.

The demonstrated susceptibility of the prickly-pears and chollas prompted a cursory examination of possible naturally affected plants. Bacilli have now been isolated several times from field collections of rotting Opuntia fulgida, O. engelmannii, and O. phaeacantha, and from soil from the saguaro forests. In culture these bacilli appear similar to E. carnegieana and, in limited tests (Table 1), have the same host-range as that organism.

DISCUSSION

Research on possible methods of controlling the bacterial necrosis of saguaro has centered in three areas: eradication, chemotherapeutic protection or treatment of affected plants, and vector control. Eradication has not proved successful, while the in vitro effectiveness of only one chemotherapeutant has thus far been demonstrated against E. carnegieana (2). Vector control has not yet proved feasible.

These approaches to control were based partly upon the assumption that the bacterial necrosis was restricted to the saguaro. The fact that other cacti, native to the saguaro range,

Table 1. Host-range of Erwinia carnegieana and of culturally similar bacilli isolated from other cacti or soil.

Species inoculated	Plant part inoc.	Saguaro isolates: Number of tests	Saguaro isolates: Number of isolates tested	Saguaro isolates: Number of plants inoc.	Saguaro isolates: % plants infected	Isolates from sources other than saguaro[a]: Inoculum source	Isolates from sources other than saguaro[a]: No. plants inoc.	Isolates from sources other than saguaro[a]: % plants infected
Carnegiea gigantea	stem	6	7	53	70	1-6 incl.	15	100
Cephalocereus mezcalaensis	stem	3	5	17	18	1,2,5	6	83
C. sartorianus	stem	2	5	14	93	1,2,5	6	83
Echinocereus engelmannii	stem	1	3	6	50	--	--	---
Ferocactus wislizenii	stem	3	5	14	100	1,2,5	3	100
Lemaireocereus thurberi	stem	3	5	13	100	5	2	100
Mammillaria microcarpa	stem	1	4	4	0	1,2,5	7	0
Opuntia engelmannii	pad	1	4	12	42	1-6 incl.	9	100
O. phaeacantha	pad	2	6	24	96	1-6 incl.	9	100
O. arbuscula	joint	1	1	4	100	1,2	4	75
O. fulgida	joint	3	6	35	74	1,2,5	3	67
O. versicolor	joint	2	4	15	53	1-6 incl.	7	86
Pachycereus grandis	stem	5	3	17	0	1,2,5	6	0
P. pecten-aboriginum	stem	5	3	17	6	1,2,5	8	0
P. pringlei	stem	5	3	17	59	1,2,5	6	50
Trichocereus spachianus	stem	5	3	17	53	1,2,5	6	67
Euphorbia obesa	stem	1	1	3	100	1,2	4	100
E. valida	stem	1	1	1	100	1,2	2	100
Cucurbita pepo var. medullosa (Italian squash)	fruit	1	4	12	42	1,2	4	100
Cucumis melo var. inodorus (honey-dew)	fruit	1	4	12	100	--	--	--
Lycopersicon esculentum (tomato)	fruit	2	4	15	100	1,2	4	100

[a]Inoculum sources: 1, 4, 6 - O. phaeacantha; 2, 3 - O. fulgida; 5 - soil.

are also susceptible suggests that a re-evaluation should be made of possible control procedures, particularly with respect to vector relationships. Confirmation of the survival in soil of bacilli similar to, if not identical with, E. carnegieana (1) suggests that this medium may be a mode for air-dispersal of this organism, in addition to its possible role in seedling infection.

Some of the susceptible cacti, such as Pachycereus pringlei and Trichocereus spachianus, are not native to the United States. However, on the lower limits of its distribution in Mexico the saguaro is associated with P. pringlei (5). Thus, it seems probable that there could be a continuation of the disease into Mexico and, possibly, into Central and South America through the medium of overlapping hosts such as species of Opuntia, Lemaireocereus, Cephalocereus, Pachycereus, and Trichocereus. Reports of bacterial rot of other cacti in these regions, however, are not known.

The obvious susceptibility of tomato and Italian squash fruits and honey-dew melons suggests that the disease could be of importance to these or similar crops (such as watermelons, 3) grown within the distribution range of E. carnegieana.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, TUCSON, ARIZONA

LOW TEMPERATURE INJURY AS A CONTRIBUTING FACTOR IN CYTOSPORA INVASION OF PLUM TREES[1]

A. W. Helton[2]

Summary

Tests were carried out under three sets of conditions to determine the most satisfactory method for local killing of stem tissues of plum trees by attached blocks of dry ice; and to determine the degree to which the killed tissues would be invaded by naturally disseminated Cytospora inoculum.

From these tests the following conclusions were reached: 1) diameter of the stem is a more determinant factor in achieving satisfactory tissue killing than size of the dry ice block; 2) covering of dry ice blocks increases tissue killing, and the kind of covering, or wrapping, is of little importance; 3) invasion of freeze-killed tissues by Cytospora fungi is easily accomplished; and 4) low temperature winter injury is, by deduction, a prime contributing factor in the development of epiphytotics of Cytospora canker.

INTRODUCTION

Cytospora fungi are efficient invaders of wounds (3). They also attack plants that have been weakened by any of several adverse conditions in the environment (4).

Contributing factors sometimes act in such subtle ways that their association with Cytospora diseases is not readily apparent, but fortunately this has not always been the case.

Although the Cytospora fungi have long been recognized as invaders of weakened tissues, they have also been known to invade healthy trees in the absence of prominent wounds or other injuries.

Fire injury was described by Povah (7) as the major contributing factor behind an outbreak of Cytospora diseases on Populus grandidentata. Drought was reported by Hubert (5) to be a weakening factor that predisposed forest trees to infection with Cytospora chrysosperma.

Wright (8) found that Populus deltoides did not develop disease when grown in good soil with adequate sub-surface moisture; whereas trees grown on hard-pan, with accompanying poor root development, were quite susceptible to Cytospora infection. He reported a parallel situation for Pseudotsuga menziesii (9).

Desiccation was noted to be a factor by Butin (1). Susceptibility was enhanced by delay in suberization accompanying increased loss of water.

Such subtle contributing factors as those involved when forest trees grow at the outer limits of their ranges were cited by Long (6). He reported 10 species of Populus and 3 of Salix as susceptible to C. chrysosperma under such circumstances. Also, that drought, cultural neglect, severe pruning, and conditions normally maintained for the rooting of cuttings were contributory to invasion by the fungus.

Forsberg (2) reported that Chinese elms were predisposed to infection by freezing temperatures, but trees growing under favorable circumstances also were attacked.

Low temperature injury is a commonly present factor in the northern latitudes, because there are few 5-year periods during which certain fruit tree species are not injured to some degree by unusually low winter temperatures or by unseasonal freezes. Severe freezes sometimes cause the bark to split on the trunks and large scaffold branches, creating ideal wounds for invasion by Cytospora fungi. Often, however, the injury is minor and such large wounds are not created. Instead, tissues are killed in small, localized spots at various locations on the injured trees.

The experiments reported here were concerned with development of a practical method of simulating localized winter-killing of stem tissues, and with the importance of localized freezing, per se, as a factor in initial infection and subsequent development of Cytospora canker diseases in plum trees in an area not devoted to commercial orcharding.

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[1]Approved by the Director of the Idaho Agricultural Experiment Station as Research Paper No. 516.

[2]Associate Plant Pathologist, Idaho Agricultural Experiment Station.

MATERIALS AND METHODS

Three groups of trees, involving three different sets of conditions, were included in the tests.

One group consisted of 1-year-old Italian prune trees (Prunus domestica) in 14-inch clay pots in a greenhouse. Having grown in the pots for 1 year, these trees were well established; they were dormant at the time of the test, having just been returned to the greenhouse from a 1°C storage room.

Another group consisted of dormant (at the time of treatment) 7-year-old Italian plot trees growing on a 10-foot grid on a gentle south slope.

The third group was composed of dormant 4-year-old Italian and Stanley prune (P. domestica) and President plum (P. domestica) trees in a plot consisting of a low central portion and moderate slopes to the south and to the north. The varieties were randomly arranged on a 6-foot grid.

A 50-pound block of dry ice was sawed into smaller blocks with a band saw a few hours before the tests were begun. Two block sizes were used in each of the three tests. In one series the blocks were 1 x 3 x 15 cm, and in the other 2 x 4 x 15 cm.

Two dry ice blocks were bound to each tree in all tests. Trunks were used in the greenhouse and in the plot of mixed plum and prune varieties. Trunk diameter averaged 1 cm in the greenhouse and 4 cm in the mixed variety plot. Branches averaging 2 cm in diameter were used in the 7-year-old Italian plot.

The blocks were attached to the test trees as soon as possible after they were sawed. They were bound to the stems with string at the middle and a nurseryman's rubber budding strip at each end, and with various coverings (on outer surface of block) and wrappings (wrapped around stem and block, except point of contact of ice and stem) between block and binding.

The coverings and wrappings were applied to determine the effects these might have in delaying evaporation of the blocks, and thereby in increasing the extent of the tissue killing in the vicinity of the blocks. All coverings and wrappings were removed as soon as the last

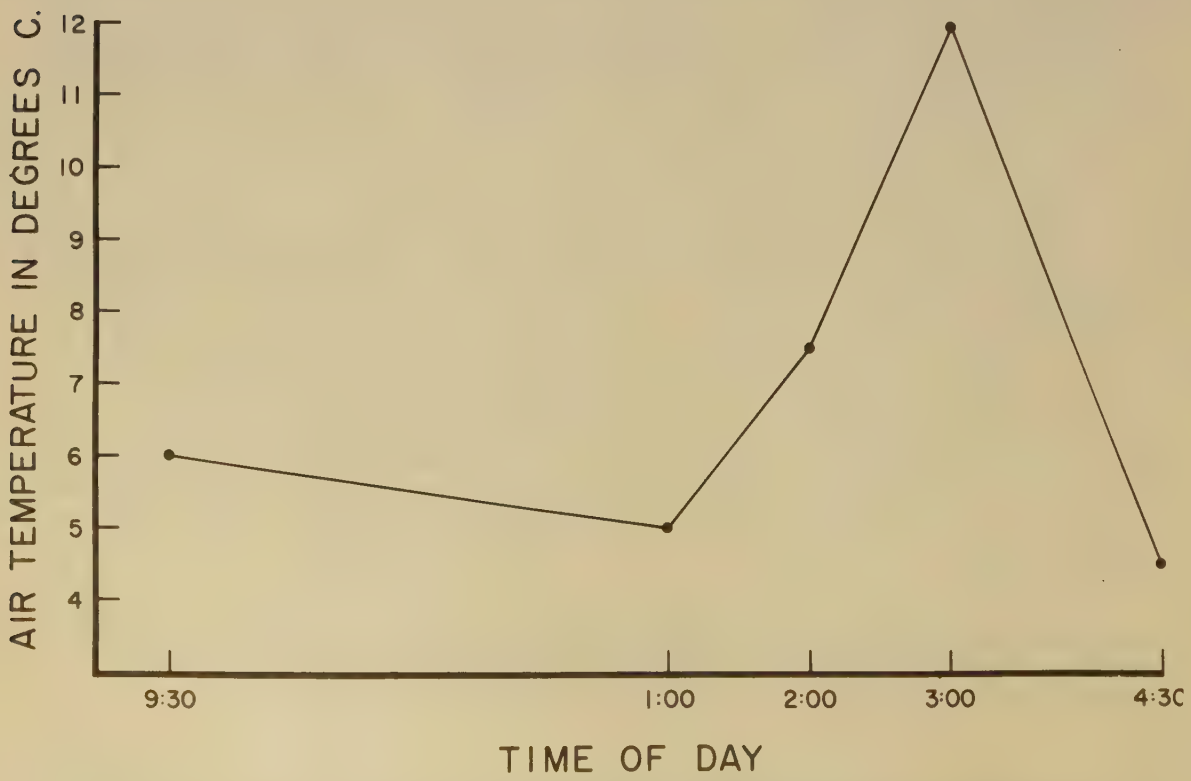


FIGURE 1. Air temperature conditions during evaporation of dry ice blocks used to freeze branches of 7-year-old Italian prune trees in a field plot in mid-March.

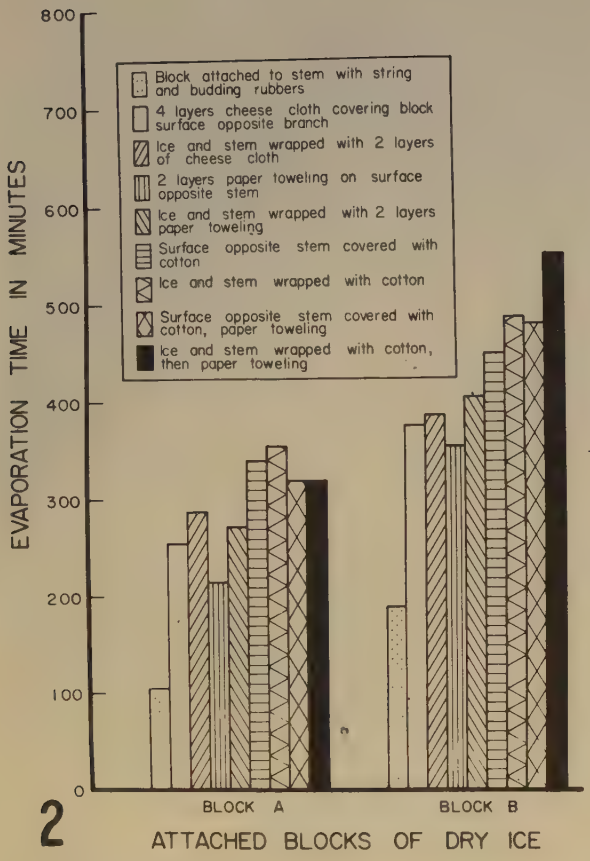
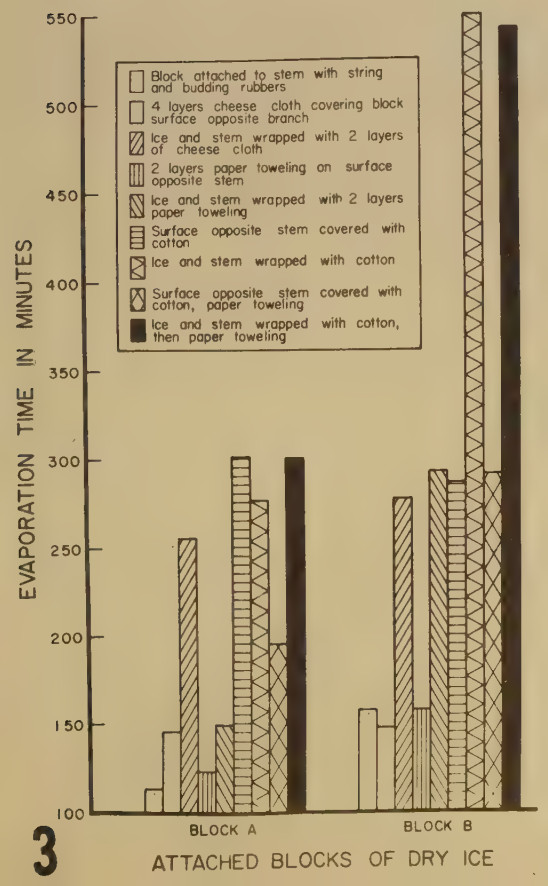
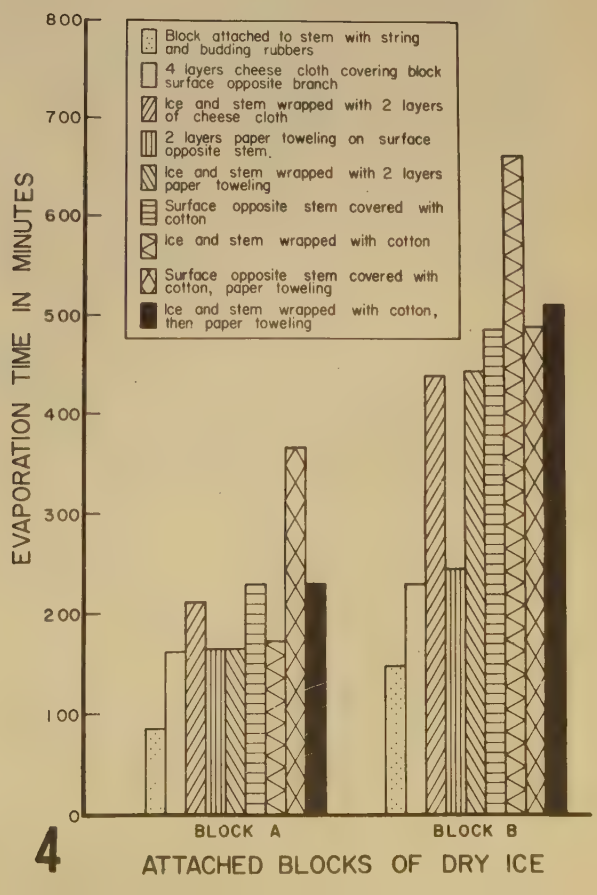

EVAPORATION TIME IN MINUTES
800
700
600
500
400
300
200
100
0
Block attached to stem with string and budding rubbers
4 layers cheese cloth covering block surface opposite branch
Ice and stem wrapped with 2 layers of cheese cloth
2 layers paper toweling on surface opposite stem
Ice and stem wrapped with 2 layers paper toweling
Surface opposite stem covered with cotton
Ice and stem wrapped with cotton
Surface opposite stem covered with cotton, paper toweling
Ice and stem wrapped with cotton, then paper toweling
BLOCK A
BLOCK B
ATTACHED BLOCKS OF DRY ICE
2

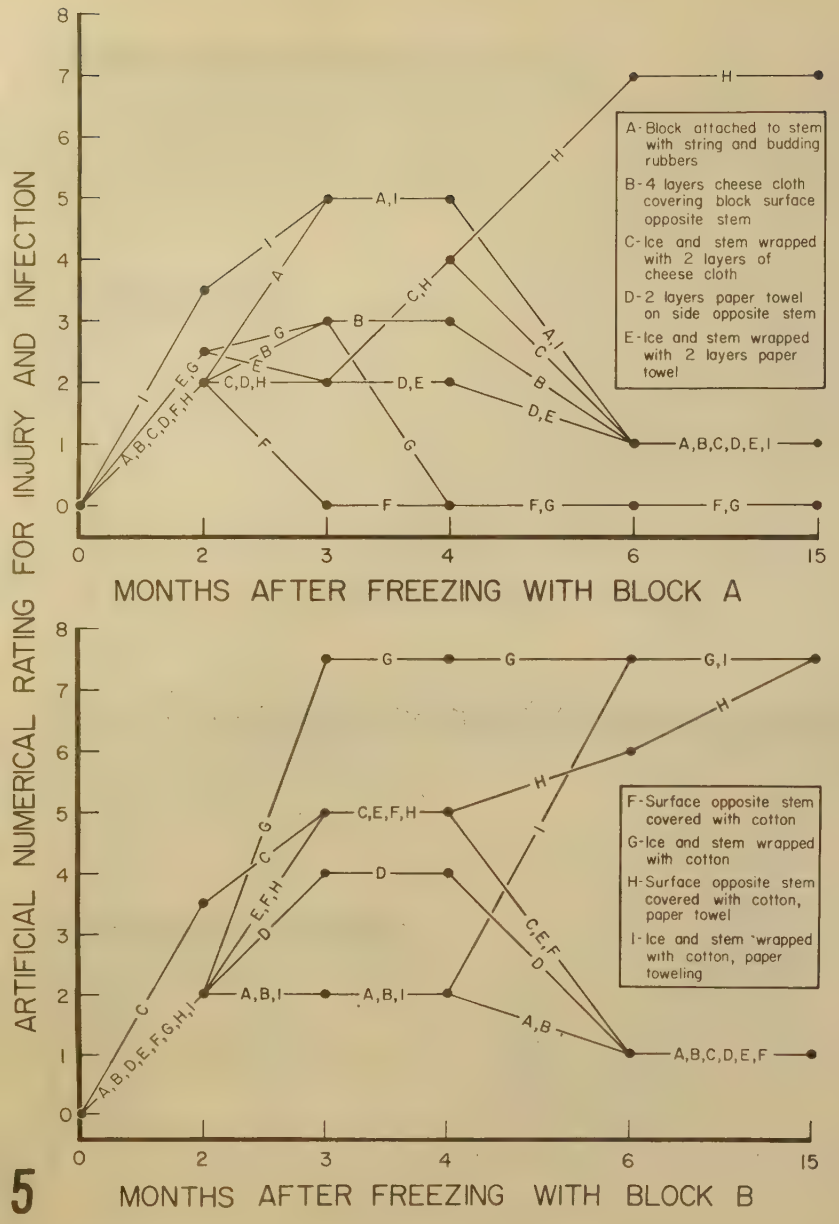
FIGURE 2. Effects of various coverings and wrappings in prolonging the life of two sizes of dry-ice blocks (A, 1 x 3 x 15 cm; B, 2 x 4 x 15 cm) when bound to trunks (approximately 1 cm in diameter) of 1-year-old Italian prune trees in a greenhouse. Average of four replicates. Average air temperature 15° C.

FIGURE 3. Effects of various coverings and wrappings in prolonging the life of two sizes of dry ice blocks (A, 1 x 3 x 15 cm; B, 2 x 4 x 15 cm) when bound to branches (approximately 2 cm in diameter) on 7-year-old Italian prune trees in a field plot in mid-March. Average of four replicates. Average air temperature 7°C.

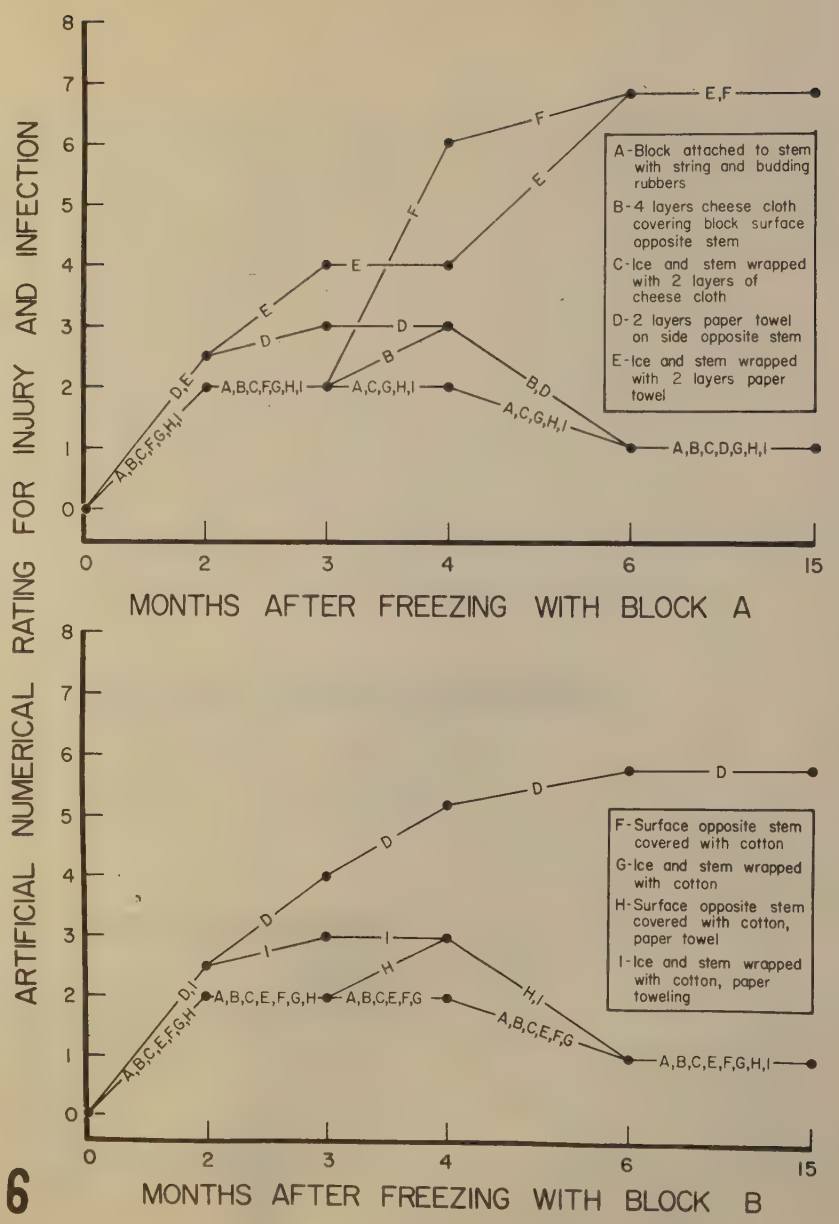
FIGURE 4. Effects of various coverings and wrappings in prolonging the life of two sizes of dry ice blocks (A, 1 x 3 x 15 cm; B, 2 x 4 x 15 cm) of 4-year-old Italian and Stanley prune and President plum trees in a field plot in mid-March. Average air temperature 7°C.


EVAPORATION TIME IN MINUTES
550
500
450
400
350
300
250
200
150
100
Block attached to stem with string and budding rubbers
4 layers cheese cloth covering block surface opposite branch
Ice and stem wrapped with 2 layers of cheese cloth
2 layers paper toweling on surface opposite stem
Ice and stem wrapped with 2 layers paper toweling
Surface opposite stem covered with cotton
Ice and stem wrapped with cotton
Surface opposite stem covered with cotton, paper toweling
Ice and stem wrapped with cotton, then paper toweling
BLOCK A
BLOCK B
ATTACHED BLOCKS OF DRY ICE
3


EVAPORATION TIME IN MINUTES
800
700
600
500
400
300
200
100
0
Block attached to stem with string and budding rubbers
4 layers cheese cloth covering block surface opposite branch
Ice and stem wrapped with 2 layers of cheese cloth
2 layers paper toweling on surface opposite stem
Ice and stem wrapped with 2 layers paper toweling
Surface opposite stem covered with cotton
Ice and stem wrapped with cotton
Surface opposite stem covered with cotton, paper toweling
Ice and stem wrapped with cotton, then paper toweling
BLOCK A
BLOCK B
ATTACHED BLOCKS OF DRY ICE
4


ARTIFICIAL NUMERICAL RATING FOR INJURY AND INFECTION
MONTHS AFTER FREEZING WITH BLOCK A
A-Block attached to stem with string and budding rubbers
B-4 layers cheese cloth covering block surface opposite stem
C-Ice and stem wrapped with 2 layers of cheese cloth
D-2 layers paper towel on side opposite stem
E-Ice and stem wrapped with 2 layers paper towel
A,B,C,D,E,I
F
F,G
F,G
MONTHS AFTER FREEZING WITH BLOCK B
F-Surface opposite stem covered with cotton
G-Ice and stem wrapped with cotton
H-Surface opposite stem covered with cotton, paper towel
I-Ice and stem wrapped with cotton, paper toweling
A,B,C,D,E,F
G,I

5


ARTIFICIAL NUMERICAL RATING FOR INJURY AND INFECTION
MONTHS AFTER FREEZING WITH BLOCK A
A-Block attached to stem with string and budding rubbers
B-4 layers cheese cloth covering block surface opposite stem
C-Ice and stem wrapped with 2 layers of cheese cloth
D-2 layers paper towel on side opposite stem
E-Ice and stem wrapped with 2 layers paper towel
E,F
A,B,C,D,G,H,I
MONTHS AFTER FREEZING WITH BLOCK B
F-Surface opposite stem covered with cotton
G-Ice and stem wrapped with cotton
H-Surface opposite stem covered with cotton, paper towel
I-Ice and stem wrapped with cotton, paper toweling
A,B,C,E,F,G,H,I
D

6

(See legend on opposite page)

block of dry ice had evaporated.

All tests were replicated four times, and eight untreated control trees were included in all tests.

The responses of treated trees, both from the standpoint of low temperature injury and subsequent natural infection, were recorded by means of an arbitrary numerical system, as follows: 0) neither a flat-face, due to local killing of tissues by the dry ice, nor evidence of infection; 1) normal marginal healing around flat-face; 2) flat-face with little evidence of marginal healing; 3) sparse development of pycnidial pustules of Cytospora fungi in the freeze-injured tissues; 4) moderate pimpling with Cytospora pustules; 5) severely pimpled; 6) exudation of gum from the freeze site and/or adjacent areas; 7) active pathogenesis, as evidenced by expanding Cytospora cankers; 8) flagged, that is, killing of parts terminal to active cankers, with foliage not abscised.

Satisfactory local killing of tissues was considered to have been achieved if growth processes between mid-March (the time of freezing) and mid-May resulted in development of a flattened area (flat-face) at each site of attachment of a dry ice block. When the 2-month observations were recorded (compare Figs. 5-6), flat faces were indicated if they had formed; but thereafter flat-faces were not recorded unless there was no other symptom or reaction.

RESULTS

All tests were begun on March 15, 1958. One day was required for evaporation of the most evaporation-resistant block-covering combinations. Air temperatures were recorded five times during the day in the 7-year-old Italian prune plot (Fig. 1). The average air temperature in this plot was 7° C. Conditions were similar in the plot consisting of 4-year-old Stanley and Italian prune and President plum trees, and the average temperature during the test period was the same. In the greenhouse the average was 15° C.

Effects of Freezing: Most of the 1-year-old trees in the greenhouse were killed above the places of attachment of the dry-ice blocks. Life of the blocks was governed by size and by type and extent of covering (Figs. 2, 5).

In the plot of 7-year-old Italian prune trees, all wrappings sustained the blocks much longer than the fully exposed control blocks, and all outer-surface coverings sustained them an appreciably longer time than the controls (Fig. 3). Areas of dead tissues approximating the size of the blocks developed where the blocks had been attached. None of the treatments were regarded as excessively damaging, as were those in the greenhouse test.

In the 4-year-old prune and plum plot, the effects of the various wrappings and coverings were much the same as they were on the 7-year-old trees insofar as prolonging block life was concerned (Figs. 4-6). Local killing of tissues occurred but only in very small spots where the blocks had been attached. This was true for the large blocks as well as for the small ones.

The tests in the greenhouse and with the 4-year-old plot trees were not considered satisfactory. In the test with 7-year-old Italian trees the larger dry ice blocks produced satisfactory branch-tissue killing in all cases, and the smaller blocks produced satisfactory branch-tissue killing in all but two cases (Fig. 5, upper). The two exceptions were treatments F and G. In these cases the flat-faces that developed were small and relatively transient, that is, the cambial injury was slight.

Natural Infection: Evidence of Cytospora invasion was observed in all of the 7-year-old Italian prune trees where the small blocks were used except D and F. The only case of progressive disease developed in treatment H, where the canker that formed caused parts terminal to it to flag, and the canker margin advanced downward into the scaffold branches (Fig. 5, upper). Invasion by Cytospora fungi occurred in all treatments where the large blocks were used except A and B. Virulent infections developed in treatments G, H, and I (Fig. 5, lower).

FIGURE 5. Effects of freezing, and resulting natural Cytospora invasion, when two sizes of dry ice blocks (A, 1 x 3 x 15 cm; B, 2 x 4 x 15cm) with various coverings and wrappings were bound to branches (approximately 2 cm in diameter) of 7-year-old Italian prune trees. Average of four replicates. Average air temperature 7° C.

FIGURE 6. Effects of freezing, and resulting natural Cytospora invasion, when two sizes of dry ice blocks (A, 1 x 3 x 15 cm; B, 2 x 4 x 15 cm) with various coverings and wrappings were bound to trunks (approximately 4 cm in diameter) of 4-year-old Italian and Stanley prune and President plum trees. Average of four replicates. Average air temperature 7° C.

In the 4-year-old plum and prune trees, both sizes of blocks caused injury to the trunks sufficient to result in discernible flat-faces. The flat-faces were so slight in most cases, however, that they were difficult to locate 2 months after freezing. (Since degree of flat-face development could not be indicated in the arbitrary recording system adopted, this inadequacy of injury is not revealed in Fig. 6.) Nevertheless, invasion by Cytospora fungi occurred in a few cases. Where the small blocks were used, four treatments resulted in development of Cytospora symptoms, and two of the infections (E and F) proved to be virulent. Where the large blocks were used, three treatments resulted in Cytospora invasion and in one case (treatment D) the infection proved to be virulent.

Neither low temperature injury nor Cytospora infection were observed in untreated control trees.

DISCUSSION AND CONCLUSIONS

The primary purpose of these experiments was to develop a practical method of accomp-- plishing local killing of stem tissues of plum and prune trees without causing obvious, and therefore severe, wounds, and without causing freeze-girdling of the stems and consequent death of branches. The secondary purpose was to determine to what extent natural infection of such localized areas would take place in a locality far removed from commercial orchards where serious Cytospora canker diseases were known to exist.

Preliminary efforts to accomplish the primary objective with carbon dioxide cylinders were not satisfactory. Too many problems were involved in handling of the cylinders, and too much time and gas were required to accomplish satisfactory tissue killing. Also, certain mechanical dangers were found to exist in releasing high pressure gas in the desired manner and location on test trees.

Cytospora fungi are of common occurrence under all environmental conditions in Idaho that permit growth of shrubs and trees, including those at high altitudes and in remote mountain localities. No evidence has been accumulated, however, that indicates what proportion of the natural inoculum in any of these environments is capable of inducing progressive disease in vigorous orchard trees.

The experiments reported here were not conducted in an orcharding locality. Because of this, because the test trees were vigorous, and because factors regarded as contributory to invasion by pathogenic Cytospora fungi were absent, the incidence of virulent infections in the test trees was not expected to be high. However, Cytospora fungi of undetermined pathogenicity characteristics were known to be present. The secondary objective was expected to be achieved, therefore, if macroscopic evidence of Cytospora invasion developed in the frozen tissues.

The greenhouse test was unsatisfactory because injury to the small trees was too severe and inoculum was not present in the environment.

The trunks of the 4-year-old trees in the field plot proved too large for satisfactory freeze-killing by dry ice blocks of the sizes used. The flat-faces produced were too small and too transient. The physical arrangement of the plot appeared to have no effect. Development of flat-faces and subsequent invasion by Cytospora fungi did not occur in the low central portion any more frequently than on the south and north slopes. The results were of interest primarily because fungus invasion occurred even when the injury was slight. Where small blocks were used, Cytospora fungi were found in the dead tissues of 44.5% of the test trees. Virulent infections developed on 22.2% of the test trees. Surprisingly, where the large blocks were used only 33.3% of the dead tissue areas were invaded, and only one case developed a virulent infection.

The primary objective was achieved with the 7-year-old trees, that is, the desired degree of low temperature injury was accomplished. The secondary objective also was achieved in that the freeze-killed tissues were invaded by Cytospora fungi with surprising frequency. Regardless of size or evaporation time of the dry ice blocks, 77.8% of all the test trees developed evidence of Cytospora invasion. However, few such cases developed progressive diseases. Where the small blocks were used only 11.1% developed virulent infections, though where the large blocks were used 33.3% became virulent.

Consideration of the data from all the tests led to the following conclusions: 1) Localized freeze-killing of stem tissues of plum and prune trees by dry ice blocks can be accomplished satisfactorily but depends as much on stem diameter as on block size, that is, 1 x 3 x 15 cm blocks are satisfactory for 2-cm stems but not for 1- or 4-cm stems. 2) Coverings or wrappings applied to dry ice blocks are not determinant factors in freeze-killing of stem tissues,

although a covering of some sort on the surfaces opposite the stems is advisable, both to protect the blocks from wind and sun and to protect the hands. 3) When dormant stems are sufficiently injured by low temperature that the injury is readily discernible from a distance of several paces after spring growth is well underway, invasion by naturally disseminated Cytospora inoculum is common. 4) Factors governing pathogenicity of Cytospora fungi subsequent to invasion of freeze-killed tissues have not been ascertained, but virulence of the fungi and vigor of the injured trees probably are foremost among them. 5) The results reported here verify the previously entertained supposition that prune trees injured by low temperatures are readily invaded by Cytospora fungi and suggest, therefore, that natural winter injury serves as a prime contributing factor in development of epiphytotics of Cytospora canker in commercial orchards, and particularly where other predispositional factors are present.

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IDAHO AGRICULTURAL EXPERIMENT STATION, MOSCOW

CONDITIONS FAVORABLE FOR ADSORPTION AND ELUTION OF TOBACCO MOSAIC VIRUS IN AN ECTEOLA-CELLULOSE COLUMN

T. E. Rawlins[1]

Success in the use of ion exchange methods for viruses apparently requires attention to numerous details in the technique. We are accordingly reporting a combination of conditions which gave excellent results with partially purified tobacco mosaic virus.

The ECTEOLA-SF cellulose used was purchased from Bio-Rad Laboratories, Richmond, California. In order to remove the finely divided ECTEOLA 3 g of the compound were placed in a 100-ml graduate and covered with about 100 ml of distilled water. The ECTEOLA was suspended in the water by agitation and then allowed to settle for about 5 minutes. The supernatant containing the finer colloidal material was poured off and discarded and water was again added to the settled ECTEOLA. This flotation process was repeated five times. The ECTEOLA was then left under water overnight in a refrigerator. It was then resuspended in water and poured into a burette. The burette (13 mm diameter) had a stop cock at the bottom and a packed glass wool plug just above the stop cock. The suspended ECTEOLA was slowly added until the column of settled ECTEOLA was about 12 cm deep. It is important that the column be under water or buffer solution at all times to prevent air from entering the column. Phosphate buffer equal to twice the volume of the column was then run through the column. This buffer was .005 M and had a pH of 7.1.

The concentrated tobacco mosaic virus (1.5 ml in the same buffer) that had been partially purified by two alternate cycles of low and high speed centrifugation was poured in above the column and was allowed to seep slowly down into the top inch of the column while this portion of the column was slowly stirred with a glass needle about 1 mm in diameter. Stirring prevents an impervious scum of virus from depositing on the upper surface of the column. A loose plug of glass wool was then added and the burette was filled with more of the same buffer. The effluent from the column was then collected as 3-ml fractions, allowing the liquid to move through the column at the rate of one drop per 15 seconds until eight fractions had been collected. Enough of the same buffer was then added to the mixing flask attached above the column to bring the volume of the buffer in the mixing flask to 50 ml. A separatory funnel was attached to the top of the mixing flask and in this was placed 50 ml of molar NaCl in .02 M phosphate buffer at pH 7.1. The flow through the column was then resumed, but at the rate of one drop per 5 seconds. During this second flow the liquid in the mixing flask was agitated by a magnetic stirrer and the NaCl-buffer from the separatory funnel began to mix with the solution in the mixing flask. Thus the concentration of NaCl solution passing through the column gradually increased.

In bright light fraction 14 showed maximum light scattering, decreasing in both directions in the adjacent fractions. The ultra violet absorption at 260 mμ was determined for the first 19 fractions. The absorption was negligible for the first 12 fractions. As shown in Table 1, the virus began to be released from the column in the 13th fraction, with a maximum amount of virus being released in the 14th and 15th fractions.

Fraction 14 showed the strong stream double refraction characteristic of virus when tested by the method of Takahashi and Rawlins[2]. From the results shown in Table 1 evidently the virus was firmly adsorbed to the column until the NaCl concentration reached approximately 0.3 molar when the virus was rapidly released from the ECTEOLA, mainly in the 14th and 15th fractions.

Table 1. Ultra-violet absorption (at 260 mμ) of fractions 11 to 19, inclusive.

Fraction number	11	12	13	14	15	16	17	18	19
Optical density	.032	.105	.50	2.22	1.84	1.02	.63	.44	.33

It is important that the denser NaCl solution should drip into the solution below it rather than being in direct contact with the solution below it. When in direct contact a denser NaCl solution will stream down into a less dense solution and will not give the gradual increase in NaCl desired.

A relatively porous ECTEOLA column appears to be best for obtaining a sharp peak with viruses.

The outlet of the mixing flask should be near the bottom of the flask in order that mixing may be complete before the NaCl solution reaches the column.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

[1]Professor of Plant Pathology, University of California, Berkeley, California.

[2]Takahashi, W. N., and T. E. Rawlins. 1933. Rod-shaped particles in tobacco mosaic virus demonstrated by stream double refraction. Science 77: 26-27.

YELLOW MOTTLE DECLINE OF COCONUTS IN THE TERRITORY OF GUAM

Otto A. Reinking[1]

INTRODUCTION

From 1910 to 1940 the Territory of Guam produced and exported 86,170,730 pounds of dried copra. In 1912 1047 tons valued at $59,924 and in 1913 565 tons valued at $37,058 were exported. Today no copra is exported; practically all coconuts are used locally primarily for livestock feed and some for human consumption. In many areas coconuts have been badly neglected and allowed to grow up in bush.

Because of the recent revived interest in agriculture and coconut production, the writer was sent to Guam in January 1961 to investigate the probable cause of the serious coconut decline and to report to the government recommendations for improving productivity and for control or eradication of the disease.

The first report of a coconut disease, which may very well have been what we are now calling yellow mottle decline, was made by Weston (10) in 1917. Dr. Weston's description of the disease certainly fits the picture as we know it today. About 10 years later (9) mention was made that the trouble might be an infectious disease.

EXAMINATION OF DECLINING COCONUTS

A preliminary survey made possible formulation of a plan for a detailed investigation of type diseased coconut areas. First inspection revealed that the disease might be identical to Cadang-Cadang disease in the Philippines (1,2,5,7). Since the most severely infested areas were in the southern half of the island, the studies were started at Merizo in the southern tip and moved northward.

A critical examination was begun of at least 10 trees in each large area of plantings where disease was in evidence. However, since typical yellow mottle decline was found in every tree at first examination, less than 10 trees were examined in most instances. The coconut areas studied are indicated in black in Figure 1 (4).

The studies were to include symptomatology of diseased trees with special reference to disease symptoms on leaves, to determine if the disease were identical with or similar to the yellow mottle decline (Cadang-Cadang) disease of the Philippines. Samples of the root system and soil of diseased and healthy trees were collected also for nematode studies conducted by Dr. John D. Radewald, Nematologist of the Pineapple Research Institute of Honolulu. These studies will be discussed under a separate heading.

Detailed Studies -- Southern Half of Island: Detailed examination of declining coconut trees in the southern and eastern coconut plantings on the island of Guam indicates that such trees are affected with the severe yellow mottle decline, a supposedly virus disease. In many of the larger coconut plantings up to 100% of the trees were diseased (Fig. 1). In other plantings some areas were still relatively free from disease and still producing. The disease, however, is present and scattered throughout the entire coconut region from Merizo to Barrigada and Agana and is a limiting factor in coconut culture.

The Malojloj-Inarajan test investigation plots were situated approximately in the center of the largest coconut plantings on the east coast. Only the area of the test, some distance from the main road, was critically examined and 100% disease found to be present. Observation from the road of the entire coconut region placed disease severity at 60% to 90%. Two trees, about 12 years old and apparently badly diseased, were selected for study along with older trees. Both of these young trees had the typical tree and leaf symptoms of yellow mottle decline (Cadang-Cadang) that are exhibited by seriously affected 40- to 50-year-old trees.

The roots of all diseased trees examined were weak, producing few secondary feeding roots, and many were red-brown throughout and dead.

Detailed Studies -- Northern Half of Island: A summary of detailed examinations on declining trees is presented in Figure 1. Disease was prevalent in all large coconut plantings from Barrigada, in the center of the Island, up the East Coast to Tarague Beach. From 60 to 100% disease was present in plantings up to Mt. Santa Rosa. Yigo is situated in the blackened area west of Mt. Santa Rosa. Spots of disease from 80 to 100% infestation were present just

[1]Special Consultant, Government of Guam.

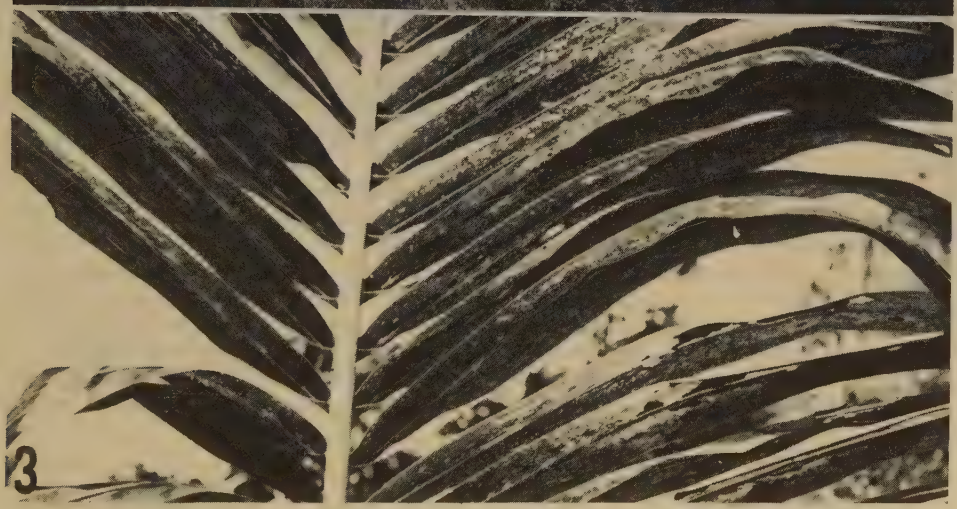
FIGURE 1. Presence and severity of yellow mottle decline. Black spots indicate largest coconut plantings.

FIGURE 2. A planting of coconuts, over 50 years old, in the last stages of yellow mottle decline.

FIGURE 3. Typical necrotic yellow mottle spotting and streaking on the lower surface of a mature leaf taken from a tree in the last stages of yellow mottle decline.

FIGURE 4. Young coconut tree, 8 to 9 years old, severely affected with yellow mottle decline. The tree is badly stunted with lower older leaves yellow bronzed, and younger, central green leaves growing abnormally, evidenced by the pasted or fan-like development.

FIGURE 5. Group of dwarfed, abnormal green nuts, some with yellow streaks and mottle, from the youngest floral group. Cocos Island.



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west of the town. In general, disease was less, north of the town with 5 to 10%, and only 2 to 5% disease north at Lupog. Young plantings on new lands opened for agriculture in the central part of the island west of Lupog and Salisbury Jct. appeared to be free from disease. Apparently the disease, as yet, has not advanced into the Tarague Beach isolated area. A cliff, some 300 feet high back from the beach, isolates the planting from coconuts planted on the mainland above. Progressing south from Tarague Beach at Finaguajac 50% disease was present. From there south, along the main highway, down to Agana practically all coconut plantings had from 60 to 100% infestation. One exception was a coastal planting on coral sands at Tumon Beach just north of Agana. Plantings boarding the beach area were diseased. In general, there was less disease in the north half of the island.

SYMPTOMS OF YELLOW MOTTLE DECLINE

The detailed symptomatology studies of coconut decline in Guam indicate that the disease is identical with or very similar to yellow mottle decline (Cadang-Cadang) in the Philippines. The symptoms of diseased trees in the Philippines and Guam were found to be identical (1,2,7). The following short description will suffice to acquaint the growers with the trouble.

General Appearances of Badly Affected Trees: Diseased trees typically show a general yellow bronzing of the older leaves, not a distinct yellowing of the foliage. Young leaves, in the center of the crown, may remain green. In advanced cases they may become somewhat yellowed and at that time small thin crowns of shortened rather upright leaves are all that is left of leaf production (Fig. 2). Old dead leaves drop off much more freely than on trees affected with a deficiency trouble. Occasionally, central green leaves fail to unfold, due to leaflets growing together, leaving an upright pasted tuft in the top of the tree (Fig. 4). The trunks of diseased trees may taper below the crown.

As the disease progresses production of nuts and normal floral parts is gradually reduced and ceases in advanced cases. Diseased nuts are small, malformed and scarified (2) with dried brown streaks (Fig. 5). Clusters of very small nuts, more numerous than normal, are produced in the last stages of nut production on badly diseased trees. Affected floral parts are at first stunted, the female flowers produce few or no nuts and in advanced cases are brown and dead. Such a tree is in severe decline, producing no nuts. Death may take place in 5 to 10 years after infection (2,6).

In severe cases, younger leaflets may be much narrowed, tough and more brittle and greener than normal; they may be malformed, broken in the middle and crinkled on the edge. A marked deterioration of the root system takes place and many dying and dead roots are present.

Individual trees respond differently to infection. In older bearing trees, the combined symptoms of decline of tree and nut and floral production along with the essential typical necrotic yellow mottle leaf symptoms make diagnosis rather simple. On young trees, not as yet in floral production, diagnosis is not so easy.

Typical Symptoms of Leaflets, Petioles, and Mid-Ribs of Badly Affected Trees: A yellow mottling that may be observed from the ground when looking through the leaflets into the sun is the most evident symptom, but close examination is essential for an accurate determination. A diseased plant cannot be diagnosed as yellow mottle decline unless typical spotting is present (Fig. 3). The true yellow mottle spotting of the disease when once recognized cannot be mistaken for spotting caused by nutritional troubles or by insects.

In yellow mottle decline the characteristic mottling, and in some cases continuous yellow to whitish streaks along both sides of the veins of leaflets, is present (Fig. 3). Typical necrotic yellow mottle spotting examined by transmitted light shows a light yellowish to whitish central translucent spot surrounded by the yellow. In reflected light the undersurface of the central part of the spot may be orange in color and has a "watery-like" appearance surrounded by a ring of yellow. Actually, spots are not watery. The center of some of the spots may be red. The spot symptoms may be observed in the fourth leaf stage and then on older leaves. The spotting is intensified on older leaves, may be very conspicuous on the leaves of trees in the last stages of the disease (Fig. 3), and remains conspicuous until the tree dies.

In addition to the typical necrotic yellow mottle, often small olive drab (so-called water-soaked) (6) spots are present on leaflets, especially on younger leaves, and also may be present on the petioles. In some cases the olive drab spotting may develop into, blotches. The symptoms are not general and may be due to a combination of causes.

CAUSE OF YELLOW MOTTLE DECLINE

The cause of yellow mottle decline (Cadang-Cadang), which was first reported by Ocfemia

(5) in 1937 in the Philippines, has as yet not been definitely established. At that time a virus was suspected as the cause. Transmission experiments started shortly after at the College of Agriculture, Los Banos, Philippines, and an almost continuous study since 1949 by scientists of the Bureau of Plant Industry, Manila and Guinobatan, Philippines, have not been successful in transmitting the disease experimentally.

During 1957-59 virologists sent by the Food and Agriculture Organization of the United Nations, Rome, to the Philippines to advise on investigations of the disease, including Drs. W. C. Price, F. P. McWhorter and Karl Maramorosch, share the opinion of most Filipino investigators that the disease is of a virus nature (3,6). Price (6) concluded that: "The evidence in favor of the virus hypothesis is very strong whereas evidence against it is very weak. So long, however, as the disease cannot be transmitted experimentally there will remain doubt that it is a virus infection." Since the writer has determined that the disease on Guam is identical with or similar to yellow mottle decline (Cadang-Cadang) in the Philippines one must be guided by the results of the studies conducted there.

RATE OF SPREAD OF YELLOW MOTTLE DECLINE

Since the disease in Guam is identical with or similar to that in the Philippines, rates of spread of the disease for Guam can only be judged by what data have been shown in the Philippines. Bigornia, Calica and Pableo from 9-year basic studies on spread indicated that the rate of spread is slow and gradual (1). The average, annual rate of spread is 4 to 6%, except in certain periods such as March to May 1952, where the average increase of incidence was 17%.

All available evidence of spread on Guam indicates that the rate is slow and gradual. If the disease actually was present by 1917, it has taken up to 43 years to wipe out the industry in certain areas like Malojloj-Inarajan. However, until a detailed survey of the coconut plantings on Guam is made, no exact data on rate of spread or extent of disease will be available. The slow spread should have a bearing on a possible rehabilitation of the coconut industry.

Seedling trees planted in the Headquarters of the Trust Territory of the Pacific Islands near Agana in 1954-55 developed a diseased condition characterized by a stunting and an abnormality in the growth and development of the youngest leaves. Fertilizer applications did not correct the trouble and in 1957 most of the diseased trees were cut down and removed. One tree of the group was left standing. On February 3 and 4, 1961 the tree showed typical symptoms of yellow mottle decline (Fig. 4).

The plantings were reported to have been made with seedlings brought from the Talofofo coconut area which is now known to have been heavily infested with yellow mottle decline. The presence of disease in the seedlings is very important from a control standpoint. If these data are accurate, the seedlings, about 3 years old, must have been diseased when planted. If true, it indicates that the disease can be transported with diseased coconut seedlings.

FERTILIZER EXPERIMENTS

Fertilizer Treatment of Abnormal Coconuts in Trust Territory Compound, Department of Agriculture, Government of Guam: At the time the tests were conducted on August 21, 1956, Dr. W. W. Cantelo, Entomologist, Department of Agriculture, treated abnormal coconuts with four different fertilizers to determine if the abnormality was due to a deficiency. Dr. Cantelo supplied the following data. Five pounds of several fertilizers was worked into soil about trees: superphosphate (3 trees), Gaviota (10-12-10) (3 trees), ammonium sulfate (3 trees), potash (3 trees). Four healthy check trees were not treated. On October 1, 1956, the trees were retreated with the same materials but not worked in. On January 22, 1957, no improvement was noted in fertilized trees, and in 1957 a number of fertilizer treated plants were removed because of disease.

On February 3, and March 3 and 13, 1961 the writer examined the one remaining diseased and fertilized tree, which had been treated with potash, and found typical leaf symptoms of yellow mottle decline. This test showed that none of the fertilizers counteracted the effects of the disease, thereby again confirming results obtained on fertilization in the Philippines (6).

Fertilizer Treatment, Malojloj-Inarajan, Department of Agriculture, Government of Guam. Data Supplied by Dr. W. W. Cantelo: During September 1956 five 50-year-old coconut trees of fairly uniform size and in approximately the same state of poor vigor were treated with five pounds each of Gaviota (10-12-10) analysis fertilizer. On February 8, 1961 all fertilized trees showed typical symptoms of yellow mottle decline. The roots were in decline, and many were dead.

Both tests, while meager, substantiate all fertilizer tests conducted in the Philippines and Guam showing that fertilizers will not correct yellow mottle decline (6).

NEMATODE INVESTIGATIONS

On March 3, 1961 a preliminary set of 24 samples of diseased roots and soil extraction specimens collected from areas of diseased and healthy trees were sent to Dr. John D. Radewald, Nematologist, Pineapple Research Institute in Hawaii. The following list of parasitic nematodes were found to be present in soils of the heavily infested Merizo and Malojloj-Inarajan coconut areas: Xiphinema spp.; Helicotylenchus nannus; Rotylenchus sp.; Aphelenchoides sp.; and Meloidogyne sp. Because of the presence of so many different parasitic genera, Dr. Radewald made the following comment: "From the samples you sent, it certainly seems that nematodes are a serious problem in the area you are in."

While these preliminary collections were small in number and no definite conclusions can be drawn as to possible relationship to coconut decline, parasitic nematodes were present, apparently in large numbers, in the 100% infested coconut plantings in the Merizo and Malojloj-Inarajan older coconut areas. A few samples collected in newly planted areas showed no parasitic nematodes.

Based on the numbers of many different parasitic genera of nematodes, especially Xiphinema spp., which are known to transmit various soil-borne viruses, Reinking and Radewald (8) suggested that yellow mottle decline may be a soil-borne virus transmitted by a Xiphinema sp.

SUGGESTED CONTROL MEASURES

1. Quarantine measures must be taken to prevent the introduction of diseased seedlings into non-diseased areas for planting. Coconut seedlings for planting new areas should be selected from trees growing in non-diseased plantings and grown in those areas before shipping out for planting.
2. Replanting plots should be made in severely infested areas to test whether economic production can be obtained for at least 10 years before disease again takes over. Data should be obtained from new plantings already made in heavily infested areas. Replanting tests should be conducted with healthy seedlings grown in healthy areas and also from selected nuts obtained from trees still growing in almost 100% infested areas.
3. Selection and breeding for resistance would be the most satisfactory means of control.

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DEPARTMENT OF AGRICULTURE, GOVERNMENT OF GUAM

RESPONSE OF ZINNIA VARIETIES TO TOBACCO RINGSPOT VIRUS[1]

J. G. Karas, D. J. deZeeuw, and C. L. Hamner[2]

Abstract

Thirty-six accessions of 32 varieties of Zinnia elegans Jacq. were evaluated for their response to tobacco ringspot virus (TRSV). The varieties differed considerably in respect to agronomic uniformity and their expression of infection. When evaluated by the appearance of mottle, chlorosis, leaf reflexing, leaf puckering, and stunting the Cupid and Lilliput types appeared most tolerant. The larger types ranged from moderately tolerant to susceptible although the virus did not produce a necrotic ring pattern and was never lethal. A progressive reduction of mottle and chlorosis, typical of TRSV, was observed in most varieties initially susceptible. One variety, "Persian Carpet" (Zinnia haageana Regel), was highly resistant.

INTRODUCTION AND LITERATURE REVIEW

Some uses of Zinnia as an investigative tool have been demonstrated comparatively recently. Price (3) first showed its utility in identification of cucumber mosaic virus strains that protect against subsequent expression of a local lesion strain. McLean (2), in a survey of tobacco ringspot virus (TRSV) diseases in the Rio Grande valley, recorded the response of Zinnia (variety omitted). The virus produced a mild chlorotic mottle from which the plants eventually recovered. Schröter (4) demonstrated the presence of alkaloids more commonly found in other plant groups. The presence of nicotine, narnicotine, and anabasine may explain graft compatibility between Zinnia and Nicotiana.

deZeeuw and Hamner have noted[3] some similarities in stunting, chlorosis, and general appearance of TRSV infected Zinnia and those treated with growth regulators such as 2,4-dichlorophenoxyacetic acid (2,4-D), naphthalene acetic acid (NAA), and indoleacetic acid (IAA). Symptomology was considered one of the means by which more basic similarities of virus and growth regulator activity could be deduced.

The value of such an investigative tool requires careful evaluation of the varietal response to both virus and growth regulator, as well as its uniformity over an extended series of experiments. This report is concerned with an evaluation of Zinnia varieties for their agronomic stability and suitability for TRSV infection in experiments of growth regulator and virus interaction.

MATERIALS AND METHODS

Seed of thirty-six Zinnia accessions representing 32 varieties were sown in the greenhouse in October. After 6 days the seedlings were transplanted to 3-inch clay pots and grown at 65° - to 70° F air temperature. No supplemental light was furnished. Plants were fed routinely with a balanced soluble fertilizer. Three-plant groups representative of the varieties in seedling condition were selected for inoculation and for controls. TRSV culture No. 3 from muskmelon (1) was used for inoculation 14 days after transplanting, at which time the plants had developed 2 to 3 pairs of leaves. Inoculation was achieved by spraying the plants at a distance of 1 inch with a 95:4:1 (vol. in ml:wt. gm.:wt. gm.) mixture of tap water, infected tobacco leaf and fine carborundum under 30 pounds' pressure in a DeVilbiss atomizer. Notes on symptom expression and on plant characteristics were made throughout the growth of the plants in the next 2 months.

Culture integrity of the virus was preserved in this and related investigations by a systematic reculturing and purification through local lesions on a TRSV-susceptible cowpea (var. Black, strain "S"). Nicotiana tabacum var. xanthi was used for growth of inoculum and routine culture of the virus.

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[2]Respectively, Departments of Horticulture, Botany and Plant Pathology, and Horticulture, Michigan State University.

[3]Unpublished notes and preliminary experiments on growth regulator-virus interactions.

Table 1. Reactions of Zinnia varieties to tobacco ringspot virus.

Variety	Healthy plants: seed source	Healthy plants: flower color[a]	Ringspot symptoms: mottle[b]	Ringspot symptoms: chlorosis[c]	Ringspot symptoms: leaf reflexing[d]	Ringspot symptoms: interveinal puckering[e]	Average dry weight per plant (gms.): healthy	Average dry weight per plant (gms.): infected	General[g] ringspot tolerance
Cupid - 1 foot tall, 1 inch flower diameter[a]									
Cupid	Burpee	Yellow	0	0	0	1	.53	.43	VT
Snowdrop	Burpee	White	1	1	3	2	--	.36	VT
Tiny Tim	Burpee	Scarlet	1	2	1	1	--	.40	T
Lilliput - 1 to 1 1/2 feet tall, 1 to 1 1/2 inches flower diameter[a]									
Canary Yellow	Burpee	Yellow	3	3	1	1.5	.50	.53	MT
Scarlet Gem	Burpee	Scarlet	1	3	2	1	--	.61	T
White Gem	Burpee	White	3	1	1	2.5	.71	.66	T
Cut and Come Again - 1 1/2 feet tall, 2 1/2 inches flower diameter[a]									
Canary Yellow	Burpee	Yellow	3	3	2	2	.77	.95	MT
Bright Scarlet	Burpee	Scarlet	1	3	1	2	.98	1.03	MT
Salmon Rose	Harris	Rose	1	2	3	3	1.10	1.21	MT
White	Burpee	White	4	3	1	3	.81	1.10	MT
Giants of California - 3 feet tall, 5 to 6 inches flower diameter[a]									
Brightness	--	Rose	3	3	4	1	.58	.46	MT
Cherry Queen	Ferry-Morse	Cherry	1	5	2	3	.97	.98	MT
Daffodil	Burpee	Yellow	5	5	5	4.5	.84	.97	MS
Enchantress	Ferry-Morse	Rose	3	3	1	2.5	1.47	1.37	MT
Lavender Gem	Ferry-Morse	Lavender	4	4	5	3	1.09	1.13	MS
Miss Wilmot	Ferry-Morse	Pink	3	5	1	3	.87	.81	MT
Orange King	Ferry-Morse	Orange	5	3	3	2	1.36	.96	MT
Purity	Burpee	White	4	4	3	2.5	.93	.87	MT
Purity	Ferry-Morse	White	3	3	4	3	.74	1.04	MT
Scarlet Flame	Ferry-Morse	Scarlet	4	5	x	3	.91	.99	MS
Scarlet King	Burpee	Scarlet	5	3	0	2	.92	1.11	MT
Violet King	Ferry-Morse	Violet	5	3	4	3	.93	.95	S
Dahlia Flowered - 3 feet tall, 5 inches flower diameter[a]									
Canary Bird	Burpee	Yellow	5	5	3	4	.80	1.17	S
Canary Bird	Ferry-Morse	Yellow	5	3	4	3	.94	1.03	MS
Polar Bear	Burpee	White	0	5	1	1	.92	1.00	MS
Will Rogers	Burpee	Scarlet-red	3	5	4	3.5	1.18	--	MS
Apricot	Burpee	Apricot	4	3	4	2.5	.96	.86	T
Blaze	Burpee (storage seed)	Orange-Scarlet	3	3	3	2	.30	.24	MS
Blaze	Burpee	Orange-Scarlet	3	2	3	2	.43	.37	MS
Burpee Gold	Burpee	Gold	3	5	3	3	.97	1.14	MT
Eskimo	Burpee	Cream-White	5	3	1	2	--	.70	--
Floradale Scarlet	Burpee	Scarlet	x[f]	x	x	x	.80	.67	S
Ice Cream	G. Ball	Cream	2	5	4	2.5	1.02	.99	MT
Tetraploid - 2 to 2 1/2 feet tall, 6 inches flower diameter[a]									
New Century	Ferry-Morse (storage seed)	Mixed	1	1	1	1	--	--	T
New Century	Ferry-Morse	Mixed	0	1	3	3	.94	1.01	T
State Fair	Ferry-Morse	Mixed	3	1	3	1.5	1.54	1.02	MT
Species - Zinnia haageana Regel - 1 foot tall, 1 inch flower diameter[a]									
Persian Carpet	Vaughn	Mixed	0	0	0	0	.14	.11	H

[a]Seedsman's description of flowers color and size, class height.
[b]Mottle ranging from 0 (no mottle) to a maximum observed 5 (relatively severe).
[c]Chlorosis ranging from 0 (no chlorosis) to a maximum observed 5 (relatively severe).
[d]Leaf reflexing ranging from 0 (relatively little) to a maximum observed 5 (severe).
[e]Interveinal puckering from 0 (none) to a maximum observed 5 (severe).
[f]x indicates apparent segregation within the variety for the character indicated.
[g]H - highly resistant, VT - very tolerant, T - tolerant, MT - moderately tolerant, MS - moderately severe, S - severe. (VS - very severe-necrosis and ringspot (not observed on Zinnia)).

RESULTS AND DISCUSSION

Table 1 summarizes characteristics of the normal plant (source, flower, color, and height variation) and the symptom range after TRSV infection (mottle, chlorosis, leaf reflexing, interveinal puckering of leaf, weight change and overall average tolerance).

Considerable variation in the characteristics evaluated is indicated and each variety has, within limits, its relatively unique reaction to TRSV. The most noticeable variation illustrated is the broad relationship of plant and flower size with susceptibility. With a few exceptions this is generally true except for the tetraploids.

In general the smallest varieties, that is Cupid and Lilliput classes, were least obviously affected by the virus, while the Dahlia flowered group was most susceptible. None of the varieties displayed any of the necrotic ring pattern commonly seen in other hosts such as tobacco and the virus was not lethal on any of the varieties.

The variety Persian Carpet was, to all intents, highly resistant, yet indicated recoverable amounts of TRSV. This additional accession is considered to be a variety of Zinnia haageana Regel and is not strictly representative of the species Z. elegans. The tolerance or immunity of Persian Carpet may be a result of natural selection since it is more nearly a wild type than others studied and may well have been subjected to the virus in its home range. Zinnia is native to regions of Mexico and southwestern United States not too distant from known infections with TRSV such as those in the Rio Grande valley (2).

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MICHIGAN STATE UNIVERSITY, EAST LANSING, MICHIGAN

THE RAPID DETERMINATION OF NECROTIC RING SPOT AND SOUR CHERRY YELLOWS VIRUSES OF STONE FRUITS[1]

R. M. Gilmer

Summary

Rapid diagnosis of necrotic ring spot virus (NRSV) or sour cherry yellows virus (SCYV) occurring either singly or in mixture in stone fruits was achieved either by a selective herbaceous host range or by single lesion transfers from cucumber cotyledons. Of these methods, the selective host range of cucumber, Butternut squash, and Tithonia speciosa permitted rapid and easy diagnosis of the two viruses, usually within 10 to 14 days.

Determination of NRSV or SCYV with stone fruit indexing hosts is always laborious and is often uncertain. Although a large number of Prunus species can be infected with either virus, the initial symptoms induced by either virus are foliar ringspots and varying degrees of necrosis (3), so that determinative diagnosis is often impossible. Montmorency sour cherry (P. cerasus) is diagnostic for SCYV in regions where temperatures of 65° F or below generally occur after bloom (4, 5), but this indicator fails to develop diagnostic symptoms of yellows in warmer areas. Peach seedlings (P. persica) are fairly reliable indicators for NRSV or SCYV in New York, but symptoms are again somewhat dependent upon temperature, and the syndromes of necrotic ring spot and sour cherry yellows often intergrade to an extent that determinative diagnosis is difficult or impossible. Either virus induces an initially localized necrotic reaction with gumming in Shirofugen flowering cherry (P. serrulata), so that the two viruses cannot be distinguished on this indicator. In any case, indexing on Prunus indicators may require from 4 to 5 weeks to 18 months before completion.

The methods contained in this report were developed as an outgrowth of large-scale indexing of numerous sweet cherry varieties (P. avium) in which diagnostic symptoms of SCYV infection do not occur. Although the methods used are, for the most part, implicit in the papers of Fulton (1, 2), the actual details of separation and diagnosis of the two viruses with herbaceous hosts appear worthy of recounting.

Virus Sources: Experiments with the selective herbaceous host range and with thermal inactivations were performed with a group of 13 isolates recovered from various stone fruits: a standard strain of NRSV (B 7/8) originally recovered from sour cherry but long carried in peach; four isolates recovered from sweet cherry seedlings infected by seed transmission; two isolates from mahaleb cherry (P. mahaleb) seedlings infected by seed transmission; four isolates from naturally infected sweet cherry orchard trees; and two isolates from naturally infected sour cherry orchard trees.

All isolates were initially transmitted from stone fruit foliage extracts to Ohio MR-17 cucumber seedlings in which the isolates were then maintained by successive transfers. The virus content of the 13 isolates and their original sources are supplied in Table 1.

Syndromes of NRSV and SCYV on the Selective Host Range: Each of the 13 isolates was transmitted from cucumber to Butternut squash (Cucurbita moschata) and to Tithonia speciosa.

Pure infections of SCYV (Table 1) were readily diagnosed on Tithonia and Butternut squash seedlings. Characteristic symptoms in these hosts are pictured in Figure 1. In Tithonia, a bright golden systemic mottle with a tendency to form rings developed in the true leaves 10 to 14 days after inoculation of the cotyledons. The mottle was prominent on the first and second leaf pairs and often decreased in prominence in later leaves. In Butternut squash seedlings, a golden systemic mottle appeared 7 to 9 days after inoculation of the cotyledons which, themselves, developed no evident symptoms. Infected plants continued to grow without apical necrosis, but their internodes were shortened and the plants were appreciably stunted. Yellow spots, blotches, or vein-banding developed in each true leaf but were less prominent or abundant in later-formed leaves. Infected plants blossomed and set fruits.

Pure infections of NRSV (Table 1) never became systemic in Tithonia plants and the virus induced only small necrotic local lesions on the inoculated cotyledons or true leaves (Fig. 2). By the time such lesions became apparent on inoculated cotyledons (4 to 5 days after inocula-

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[1]Approved by the Director as Journal Paper No. 1267, New York State Agricultural Experiment Station, Geneva.

Table 1. Virus content of experimental isolates recovered from various stone fruits.

Isolate	Virus content	Original host
B 7/8	NRSV	cerasus
2-1	NRSV	avium
1-1	NRSV + SCYV	cerasus
1-3	NRSV + SCYV	cerasus
4-4	NRSV + SCYV	avium
K-10[a]	NRSV + SCYV	mahaleb
K-45[a]	NRSV + SCYV	mahaleb
4-41[a]	NRSV + SCYV	avium
4-57[a]	NRSV + SCYV	avium
KMY[a]	SCYV	avium
KHY[a]	SCYV	avium
1-5	SCYV	avium
20-24	SCYV	avium

[a]Isolates obtained from seedlings infected by seed transmission.

Table 2. Separation of NRSV and SCYV from a mixed infection in cucumber by local lesion transfer.

Single lesion isolate[a]	Reaction on: Tithonia	Butternut	Virus content
1-3 A	-[b]	NRSV	NRSV
B	-	NRSV	NRSV
C	mottle	NRSV	NRSV + SCYV
D	-	NRSV	NRSV
E	mottle	mottle	SCYV
F	mottle	NRSV	NRSV + SCYV
G	-	NRSV	NRSV
H	-	NRSV	NRSV
I	mottle	mottle	SCYV
J	-	NRSV	NRSV

[a]The original isolate (1-3) was NRSV + SCYV.
[b]A dash indicates no systemic symptoms.

Table 3. Isolation of NRSV from mixed infections with SCYV by heat treatment.

Isolate	Initial virus content[a]	Reaction after heat treatment: Tithonia	Butternut
B 7/8	NRSV	-[b]	NRSV
2-1	NRSV	-	NRSV
1-1	NRSV + SCYV	-	NRSV
1-3	NRSV + SCYV	-	NRSV
4-4	NRSV + SCYV	-	NRSV
K-10	NRSV + SCYV	-	NRSV
K-45	NRSV + SCYV	-	NRSV
4-41	NRSV + SCYV	-	NRSV
4-57	NRSV + SCYV	-	NRSV
KMY	SCYV	-	0[c]
KHY	SCYV	-	0
1-5	SCYV	-	0
20-24	SCYV	-	0

[a]Determined by assay on Tithonia and Butternut controls.
[b]A dash indicates no systemic golden mottle developed.
[c]0 indicates no infection.

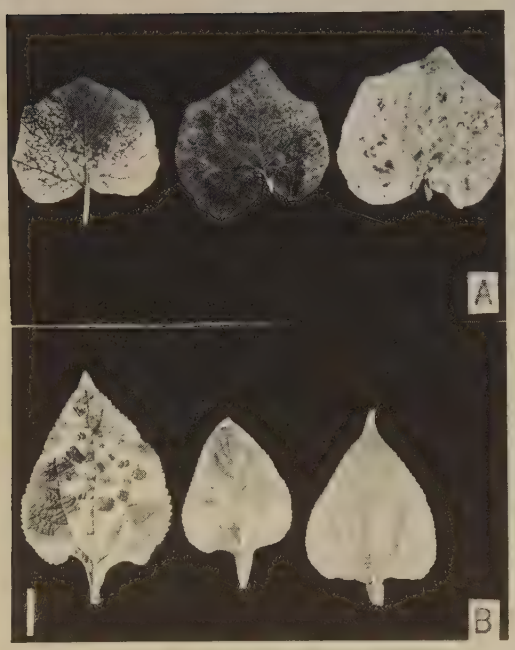
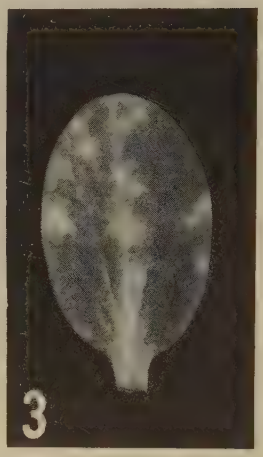


FIGURE 1. Symptoms of SCYV: A) in Butternut squash B) in Tithonia speciosa (leaf on right is healthy).

FIGURE 2. Local lesions of NRSV on inoculated leaf of Tithonia.

FIGURE 3. Chlorotic lesions of NRSV on inoculated cotyledons of Butternut squash.



tion), NRSV could be recovered from the cotyledons only occasionally, but the virus could be recovered fairly readily from inoculated cotyledons prior to the development of visible lesions. NRSV was never obtained from the true leaves of inoculated Tithonia in any of six attempts. In Butternut squash seedlings, pure NRSV infections were first evident as fairly large (3 to 5 mm in diameter) chlorotic lesions (Fig. 3) that developed on the inoculated cotyledons within 5 to 7 days. These lesions often became necrotic with age or under conditions of short day-length. If very young plants were inoculated, necrosis of the growing point often occurred before any true leaves developed, and infected plants persisted for a week or more with only their cotyledons. More commonly, when slightly older plants were inoculated, the first true leaf had partially expanded before systemic invasion of the virus killed the growing point. In such cases, the true leaf did not expand to normal size and was variously twisted with prominent semi-necrotic areas (Fig. 4). Infected plants often persisted in the 1-leaf stage for 3 or 4 weeks, but they never flowered or fruited.

Mixtures of NRSV + SCYV (Table 1) generally induced symptoms in Butternut squash seedlings that could not be distinguished from those of NRSV alone (Fig. 4), although a rare plant sometimes developed the bright golden mottle of SCYV. But the presence of SCYV in dually infected Butternut plants could be determined by transmissions to Tithonia where the golden systemic mottle induced by SCYV became apparent.

Separation of the Viruses by Local Lesion Transfer: Although NRSV and SCYV were individually separated from mixtures of the two viruses by local lesion transfers from cucumber cotyledons, the technique was laborious and often uncertain. Pure isolates of NRSV were obtained with some frequency, but isolation of pure SCYV required the transfer of a fairly large number (10 to 20) of lesions.

Sap inoculum prepared from cotyledons of cucumber plants with mixed infection was diluted with .06 M K_2HPO_4 buffer to an approximate dilution of 1:50-1:75 and was rubbed on the cotyledons of healthy Ohio MR-17 plants. This concentration of inoculum generally resulted in the development of 1 to 5 lesions per cotyledon. Individual well-isolated lesions were removed with a 4-mm cork-borer and provided inoculum to infect additional cucumber plants. Following the establishment of single-lesion isolates in cucumber, their virus content was assayed on Butternut squash and Tithonia seedlings. Results of one such experiment are given in Table 2.

On Ohio MR-17 cotyledons, local lesions of NRSV commonly appeared within 3 to 4 days after inoculation as small (1 to 2 mm diameter) rather sharply defined chlorotic spots. Local lesions of SCYV generally appeared 1 to 3 days later (4 to 7 days after inoculation) as somewhat larger (3 mm diameter) spots with rather diffuse margins. Although these differences in time of development and appearance were fairly consistent, they were not sufficiently pronounced to permit concise diagnosis of the virus content of individual lesions without assay on Tithonia and Butternut seedlings.

NRSV lesions were much more abundant than SCYV lesions in diluted inoculum from a mixture of the two viruses, being, in general, 7 to 8 times as numerous as those of SCYV.

Separation of NRSV from Mixed Infections by Heat Inactivation: Cucumber sap inoculum from each of the 13 isolates was subjected to heat at 50° ± 1°C for 10 minutes. This temperature exposure is sufficiently high to inactivate SCYV (Fulton's virus B) without inactivating NRSV (Fulton's virus G) (2). Even though the temperature is 4° to 6° below the thermal inactivation point of NRSV, it considerably lowered the titre of NRSV. Symptoms of infection in cucumber seedlings inoculated with heat-treated sap were usually delayed until 10 to 12 days after inoculation, although plants inoculated with non-heated control sap developed symptoms within the usual 4 days. Seedlings infected with heat-treated inoculum were assayed on Butternut and Tithonia seedlings to determine their virus content. In no case (Table 3) was SCYV recovered.

Separation of the Constituent Viruses from Artificial Mixtures: Two isolates of NRSV (B 7/8 and 2-1) were mixed with two isolates of SCYV (1-5 and KHY) in the four possible combinations. Equal aliquots of sap inoculum of the isolates were prepared from infected cucumber cotyledons, thoroughly mixed, and the resultant combined inoculums used to inoculate healthy Ohio MR-17 cotyledons. Shortly after local lesions developed on these cotyledons, entire cotyledons were assayed on Tithonia and Butternut squash. Both viruses were recovered. Isolates 1-5 and KHY were then recovered from infected Tithonia plants and isolates B 7/8 and 2-1 were recovered by the method of heat inactivation of SCYV indicated previously.

FIGURE 4. A) Symptoms of a mixture of NRSV +SCYV in Butternut squash B) Symptoms of NRSV in the same host.

DISCUSSION

As indicated in the introduction, the methods of separating NRSV and SCYV reported here are implicit in the data previously presented by Fulton (1, 2). The present procedures are merely a practical application to permit rapid and easy diagnosis of SCYV and NRSV from field infections in stone fruits. Of the various methods recounted, analysis on Butternut and Tithonia seedlings is by far the most simple and satisfactory. In brief, the virus or virus mixture is initially transmitted from stone fruit foliage extracts to cucumber seedlings and thence to Tithonia and Butternut squash seedlings. Pure SCYV isolates induce a bright golden systemic mottle of both Tithonia and Butternut squash. Pure NRSV isolates induce apical necrosis, large chlorotic local lesions of the cotyledons, and semi-necrotic leaf lesions of Butternut squash seedlings but do not induce systemic infection of Tithonia seedlings. Mixtures of NRSV +SCYV induce apical necrosis, local cotyledonary lesions, and semi-necrotic leaf lesions of Butternut squash seedlings and bright golden systemic mottle of Tithonia seedlings. As a routine precaution, at least four plants each of Tithonia and Butternut squash should be used to assay each virus isolate in order to diminish the possibility of false negative reactions.

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DEPARTMENT OF PLANT PATHOLOGY, NEW YORK STATE AGRICULTURAL EXPERIMENT STATION. GENEVA

THE FREQUENCY OF NECROTIC RING SPOT, SOUR CHERRY YELLOWS, AND GREEN RING MOTTLE VIRUSES IN NATURALLY INFECTED SWEET AND SOUR CHERRY ORCHARD TREES[1]

R. M. Gilmer

Summary

Of 95 sweet cherry orchard trees, ranging in age from 7 to 25 years, 49 were infected with necrotic ring spot virus (NRSV) or sour cherry yellows virus (SCYV). Of the 49 infected trees, 21 were infected with NRSV, 10 were infected with SCYV, and 18 were infected with both viruses. At least 5 trees free of NRSV and SCYV were infected with green ring mottle virus (GRMV); none of these trees developed foliage or fruit symptoms indicative of infection.

In a comparable group of 37 sour cherry trees, ranging in age from 13 to 27 years, 33 were infected with NRSV or SCYV. Of the 33 infected trees, 5 were infected with NRSV, 2 were infected with SCYV, and 26 were infected with both viruses. None of the sour cherry trees were infected with GRMV.

When grouped into age classes, 16 of 42 sweet cherry trees aged 21 to 25 years were free of NRSV and SCYV; but of 28 sour cherry trees in a similar age class (21 to 27 years old) only 1 was free of NRSV and SCYV.

These data suggest that the spread of NRSV and SCYV is much less rapid in sweet cherry orchards than in sour cherry orchards in New York.

The frequency and the rates of spread of NRSV, SCYV, and GRMV in sour cherry orchards in eastern North America have been studied by several workers (1, 4, 5, 6). Surveys for the occurrence of NRSV and SCYV and for rates of spread of these viruses have been made in Ontario (6), New York (4), and Pennsylvania (5), and, in spite of differences in spread rates from these three areas, the data presented are quite similar. These data indicate a very rapid spread of NRSV in sour cherry orchards between the fourth and tenth years after planting, and a somewhat slower spread of SCYV which commences to assume significant proportions 8 to 10 years after planting. Similar surveys for the spread of GRMV have been made by Barksdale (1) and indicate that this virus spreads fairly slowly in New York sour cherry orchards.

Although all three viruses occur naturally in sweet cherry, very little is known of their frequency or their rates of spread in sweet cherry orchard trees. Symptoms of GRMV and SCYV infections in sweet cherry are obscure or masked, so that visual surveys for such infections are very unsatisfactory.

In the course of a search for virus-free propagating materials of several sweet cherry varieties, a number of trees in a sweet cherry variety collection were indexed. This report deals with an analysis of the viruses found in certain of these trees and in a companion sour cherry orchard.

EXPERIMENTATION

The sweet cherry trees analyzed for virus content were in a mixed collection of sweet and sour cherry varieties planted in 1936. Because of the removal of certain of the original trees, many of the trees sampled were replants of various ages, but all samples were obtained from trees planted before 1954.

A total of 112 trees from this orchard were indexed: 17 trees of 8 sour cherry varieties and 95 trees of 45 sweet cherry varieties. In addition, 20 trees from an adjacent Montmorency sour cherry orchard planted in 1934 were indexed. The Montmorency orchard had been visually surveyed for the occurrence of sour cherry yellows annually from 1946 through 1959.

All trees were initially indexed on cucumber seedlings in 1957 and 1958 by the foliage juice extract technique (3). Following a third indexing on cucumber in 1959, all of the apparently healthy trees (cucumber-negative) were further indexed by bud-inoculation to Shirofugen flowering cherry (Prunus serrulata) during the summer of 1959. Trees that did not index positive on cucumber or Shirofugen cherry were not tested further, although certain of them later proved to be infected with GRMV.

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Following virus transfers from cherry foliage juice extracts to cucumber seedlings in 1960, individual virus isolates were transmitted from cucumber to seedlings of Butternut squash (Cucurbita moschata) and to seedlings of Tithonia speciosa. The use of Tithonia permitted the separation of SCYV from mixtures of NRSV + SCYV, and heat inactivation (50° C ± 1° for 10 minutes) of SCYV permitted the separation of NRSV from mixtures of NRSV + SCYV (2). The use of differential herbaceous hosts and heat inactivation thus made possible the determination of the virus content in both sweet and sour cherry foliage.

RESULTS

No indications of the transmission of GRMV to herbaceous hosts were obtained in any of these experiments and there were no indications of the presence of this virus in the initial exploratory experiments in 1957 and 1958. GRMV, however, induced leaf curl and veinal necrosis (compare 1, 3) in Shirofugen indicators in 1960 after bud-inoculation in 1959. Because of the type of indexing technique used -- in which five presumably healthy sweet cherry trees (cucumber-negative) were indexed on individual branches of a single large Shirofugen tree -- the frequency of GRMV is not accurately known. Of 46 presumably healthy sweet cherry trees thus indexed in groups of five or six on nine Shirofugen indicators, a minimum of five were infected with GRMV since five of the Shirofugen indicators developed symptoms of green ring mottle. GRMV may also have been present without detection in sweet cherry trees infected with NRSV or SCYV, since, with rare exceptions, these latter viruses were detected on cucumber and the carrier trees were consequently not indexed on Shirofugen.

GRMV induces distinctive foliage and fruit symptoms in sour cherry (1), and the absence of symptoms in the sour cherry trees sampled was relied upon as indicating absence of this virus. No symptoms of GRMV were observed in any of the 37 sour cherry trees from 1957 through 1960.

The results of the general indexing on cucumber are given in Table 1, and the virus analysis of the infected trees in Table 2.

Rates of Spread of NRSV and SCYV in the Sour and Sweet Cherry Orchards: The incidence of NRSV and SCYV in the mixed sweet and sour cherry variety collection orchard and in the adjacent Montmorency orchard at the time these two orchards were planted (1936 and 1934, respectively) is not known, but it can be safely assumed that some proportion of the original trees planted in both orchards was infected at the time of planting.

Visual surveys for the occurrence of sour cherry yellows in the Montmorency orchard were initiated in 1946[2], when the orchard was 12 years of age, and were continued annually through 1959, at which time 94% of the trees had become infected with SCYV. The rapid rate of spread of SCYV in this orchard is presented in Table 3. Although no data were obtained on the rate of spread of NRSV, it is obvious from the virus analysis of the 20 trees sampled (Table 2) that a high proportion of the trees are infected with this virus. Indexing data from 1947 supplied by K. G. Parker indicate that essentially all trees still free of sour cherry yellows at that time were already infected with NRSV.

Since visual surveys for the spread of yellows in sweet cherry were not possible, no similar data on the spread of SCYV in sweet cherries are available. During the period of intensive indexing of sweet cherry from 1957 to 1960, only two previously healthy sweet cherry trees became infected. When the indexing data are considered on the basis of tree age (Table 4), it is obvious that the spread of NRSV or SCYV in sweet cherry has been much less rapid than in sour cherry trees of comparable age. Of the 42 sweet cherry trees in the 21 to 25 year age class, 16 are still free of either NRSV or SCYV.

DISCUSSION

The high incidence of NRSV and SCYV in the Montmorency planting is quite characteristic of eastern sour cherry orchards, and the rapid spread of SCYV that occurred after the trees were 12 years of age is not at all unusual (compare 4, 6). A Montmorency orchard tree, 15 years old or older, that is still free of NRSV or SCYV is a rarity in the eastern United States.

In contrast, a sizeable number -- 16 of 42 trees (38%) -- of sweet cherry trees in the 21 to 25 year age class are still free of either NRSV or SCYV. In the infected sweet cherry trees, single infections of NRSV or of SCYV were common, although single infections of either of these viruses were relatively uncommon in the sour cherry trees analyzed (Table 2). These data indicate that NRSV and SCYV do not spread as rapidly in sweet cherry as they do in sour cherry

[2]Surveys in this orchard from 1946-1950 were made by K. G. Parker, who kindly permitted the use of his data.

Table 1. Incidence of NRSV and SCYV in sweet and sour cherry orchard trees at Geneva, New York.

Cherry species	Number of trees indexed	Number of trees infected	% of trees infected
Sweet cherry	95	49	52
Sour cherry[a]	37	33	89

[a] Sample consisted of 17 trees of 8 varieties interplanted in the sweet cherry orchard plus 20 trees of Montmorency from a planting about 300 yards away.

Table 2. Analysis of NRSV and SCYV content of sweet and sour cherry orchard trees.

Cherry species	Number of trees analyzed	Number of trees infected with:		
		NRSV	SCYV	NRSV + SCYV
Sweet cherry	49	21	10	18
Sour cherry	33	5	2	26

Table 3. Spread of sour cherry yellows in Montmorency sour cherry, Geneva, 1946-1958.

Year	Number trees infected/Number trees surveyed	% of trees infected
1946[a]	42/117	36
1950	51/117	44
1954	82/110[b]	74
1958	101/107[b]	94

[a] The trees, planted in 1934, were 12 years of age at the first survey.
[b] The decrease in number of trees surveyed was caused by tree removal.

Table 4. Incidence of NRSV and SCYV in sweet and sour cherry orchard trees by age class.

Age class[a] (years)	Number of trees indexed	Number of trees infected	% of trees infected
Sweet cherry[b]			
21 - 25	42	26	62
10 - 15	16	8	50
6 - 9	24	10	42
Sour cherry			
20 - 27	28	27	96
16 - 19	3	3	100
11 - 15	6	3	50

[a] In 1960
[b] Planting records of 13 indexed trees did not permit placement into a definite age class; these trees are omitted from the data presented.

under conditions in western New York. The possible reasons for this difference in the rate of spread of these viruses in the two cherry species are obscure.

Although the frequency of occurrence of GRMV in sweet cherry is not definitely established by the data presented, certainly this virus is more common in sweet cherry in New York than previously supposed. Careful visual examination of the possible infected sweet cherry trees failed to detect any leaf or fruit symptoms indicative of infection. The economic significance of green ring mottle in sweet cherry is not known.

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DEPARTMENT OF PLANT PATHOLOGY, NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, GENEVA

PYTHIUM ULTIMUM AND HELMINTHOSPORIUM VAGANS AS FOLIAR PATHOGENS OF GRAMINEAE[1]

Laurence D. Moore and Houston B. Couch[2]

Summary

Pythium ultimum Trow. was found to be capable of inciting foliar blighting of 45 species, representing 21 genera, of Gramineae. Thirty-five of these species have not previously been reported susceptible to the pathogen. With the exception of big bluestem (Andropogon gerardi Vitman), all species of Gramineae tested that had previously been reported susceptible to P. ultimum were confirmed. All common turfgrass species tested, except for tall fescue (Festuca arundinacea Schreb.) and roughstalk bluegrass (Poa trivialis L.), were found to be susceptible to the organism.

Thirty species from 18 genera of Gramineae were susceptible to Helminthosporium vagans Drechs. Only one of these species had previously been reported susceptible to the pathogen. Although previously reported as a host, orchardgrass (Dactylis glomerata L.) was not parasitized by H. vagans. Of the common turfgrass species tested, tall fescue (Festuca arundinacea Schreb.), common ryegrass (Lolium multiflorum Lam.), perennial ryegrass (L. perenne L.), big bluegrass (Poa ampla Merr.), annual bluegrass (P. annua L.), Kentucky bluegrass (P. pratensis L.), and roughstalk bluegrass (P. trivialis L.) were parasitized. Of the total group, those most susceptible to H. vagans were Johnsongrass (Sorghum halepense (L.) Pers.), sorghum (S. vulgare Pers.), and Sorghum almum L.

Pythium blight, incited by Pythium aphanidermatum (Edson) Fitzpatrick (Syn. P. butleri Subrm. (8)) and P. ultimum Trow., and Helminthosporium melting-out, incited by Helminthosporium vagans Drechs., are among the most important diseases of turfgrass.

Initially, Pythium blight was recorded occurring on creeping bentgrass (Agrostis palustris Huds.) in the north-central, northeastern and southern sections of the United States (9). The malady has since been erroneously typified as a disease of southern-grown turfgrass. In reality, while not as common as in the South, Pythium blight, particularly that incited by P. ultimum, is consistently an important disease in the northern States (10).

Although Pythium spp. are commonly considered to be root, crown, and seedling pathogens, foliar blighting is the primary economic loss associated with their parasitic activity on turfgrass. As a result, it is difficult to utilize literature relative to host ranges (4, 8, 12) of P. aphanidermatum and P. ultimum in predicting their capabilities for foliar blighting on various species of Gramineae.

Helminthosporium melting-out was first reported in 1919 when it was recognized on Kentucky bluegrass (Poa pratensis L.) (3). Considered a minor problem at first, melting-out has since been recognized as one of the most important diseases of Kentucky bluegrass in the northeastern section of the United States. Once the disease becomes established in a turfgrass stand, it remains an ever-present problem. In the cool humid months of spring, incidence of infection is often so high that the leaves are girdled and drop from the plants.

In addition to Kentucky bluegrass, H. vagans has been reported on orchardgrass (Dactylis glomerata L.), meadow fescue (Festuca elatior L.), prairie Junegrass (Koeleria cristata (L.) Pers.), plains bluegrass (Poa arida Vasey), bulbous bluegrass (P. bulbosa L.), Canada bluegrass (P. compressa L.), Sandberg bluegrass (P. secunda Presl), and roughstalk bluegrass (P. trivialis L.) (1,2,6,11).

In the present investigation, the potential of P. ultimum as a foliar pathogen was tested on 78 species representing 33 genera of Gramineae. Seed of these various species, obtained from Dr. A. A. Hanson of the Forage and Range Branch of the United States Department of Agriculture, was sown in 6-inch plastic pots with a soil mixture as the supporting medium. Hitchcock's manual (5) and the Kew publications (7) were used as the authorities for all plant species designations.

To determine whether conditions were suitable for disease development, Highland bentgrass,

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[2]Research assistant and Associate Professor of Plant Pathology, respectively.

Table 1. Susceptibility of species of Gramineae to Pythium ultimum and Helminthosporium vagans.

Scientific name	Common name	Susceptibility	
		P. ultimum	H. vagans[a]
Agropyron cristatum (L.) Gaertn.	crested wheatgrass	S	NS
Agropyron desertorum (Fisch.) Schult.	desert wheatgrass	S	S
Agropyron inerme (Scribn. & Smith) Rydb.	beardless wheatgrass	S	S
Agropyron intermedium (Host) Beauv.	intermediate wheatgrass	--	S
Agropyron pungens (Pers.) Roem. & Schult.		--	NS
Agropyron rigidum Beauv. (A. elongatum Host)	tall wheatgrass	S	S
Agropyron riparium Scribn. & Smith	streambank wheatgrass	S	NS
Agropyron sibiricum (Willd.) Beauv.	Siberian wheatgrass	--	NS
Agropyron smithii Rydb.	bluestem wheatgrass	S	NS
Agropyron trachycaulum (Link) Malte x Hordeum jubatum L.		S	--
Agropyron trichophorum (Link) Richt.	stiffhair wheatgrass	S	NS
Agrostis alba L.	red top	S	NS
Agrostis palustris Huds.	seaside bentgrass	S	--
Agrostis tenuis Sibth.	Colonial bentgrass	S	NS
	Highland bentgrass	S	--
Alopecurus arundinaceus Poir.		NS	--
Alopecurus pratensis L.	meadow foxtail	S	NS
Andropogon gerardi Vitman (A. furcatus Muhl.)	big bluestem	NS	--
Andropogon hallii Hack.	sand bluestem	NS	NS
Andropogon ischaemum L.	turkestem	NS	NS
Andropogon nodosus (Willem.) Nash (A. annulatus Forsk.)	Angletongrass	NS	S
Andropogon saccharoides Swartz	silver beardgrass	--	NS
Andropogon scoparius Michx.	little bluestem	NS	S
Arrhenatherum elatius (L.) Presl	tall oatgrass	S	NS
Axonopus affinis Chase	common carpetgrass	S	--
Bouteloua curtipendula (Michx.) Torr.	side-oats grama	S	NS
Bouteloua eriopda (Torr.) Torr.	black grama	--	NS
Bouteloua gracilis (H. B. K.) Lag.	blue grama	--	NS
Bromus arvensis L.	field brome	S	NS
Bromus catharticus Vahl	rescuegrass	NS	--
Bromus inermis Leyss.	smooth brome	S	NS
Bromus marginatus Nees		--	NS
Bromus mollis L.	soft brome	S	--
Bromus valdivienus Phil.		NS	--
Buchloë dactyloides (Nutt.) Engelm.	buffalograss	NS	S
Chloris gayana Kunth	Rhodesgrass	NS	S
Cynodon dactylon (L.) Pers.	Bermudagrass (common)	S	--
	Tiflawn Bermudagrass	NS	--
	U-3 Bermudagrass	NS	--
Cynodon dactylon x C. transvaalensis Davy	Tifgreen Bermudagrass	NS	--
Dactylis glomerata L.	orchardgrass	S	NS
Digitaria eriantha Steud.		--	NS
Echinochloa crusgalli var. frumantacea (Roxb.) W. F. Wight	Japanese millet	--	S
Ehrharta calycina J. E. Smith		--	NS
Elymus canadensis L.	Canada wild-rye	S	--
Elymus giganteus Vahl		--	S
Elymus junceus Fisch.	Russian wild-rye	S	S
Eragrostis bicolor Nees		--	NS

Table 1. (continued)

Scientific name	Common name	Susceptibility[a] P. ultimum	Susceptibility[a] H. vagans
Eragrostis chloromelas Steud.	Boer lovegrass	S	NS
Eragrostis curvula (Schrad.) Nees	weeping lovegrass	S	NS
Eragrostis lehmanniana Nees	Lehmann lovegrass	S	NS
Eragrostis superba Peyr.		S	--
Eragrostis trichodes (Nutt.) Wood	sand lovegrass	NS	NS
Eremochloa ophiuroides (Munro) Hack.	centipedegrass	NS	--
Festuca arundinacea Schreb.	tall fescue	NS	S
Festuca elatior L.	meadow fescue	S	--
Festuca ovina L.	sheep fescue	--	NS
Festuca rubra L.	red fescue	S	--
Hilaria jamesii (Torr.) Benth.	galleta	--	NS
Holcus lanatus L.	velvetgrass	NS	NS
Hordeum bulbosum L.	bulbous barley	NS	NS
Lolium multiflorum Lam.	common ryegrass	S	S
Lolium perenne L.	perennial ryegrass	S	S
Lolium subulatum Vis.	wimmera ryegrass	S	--
Muhlenbergia wrightii Vasey	spike muhly	--	S
Oryzopsis hymenoides (Roem. and Schult.) Ricker	Indian ricegrass	NS	NS
Oryzopsis miliacea (L.) Benth. and Hook.	smilograss	S	NS
Panicum antidotale Retz.	blue panic	S	S
Panicum virgatum L.	switchgrass	S	S
Paspalum dilatatum Poir.	Dallisgrass	NS	--
Pennisetum ciliare (L.) Link		NS	NS
Pennisetum glaucum (L.) R. Br.	pearl millet	NS	S
Phalaris arundinacea L.	reed canarygrass	NS	NS
Phalaris coerulescens Desf.		NS	NS
Phalaris minor Retz.		NS	--
Phalaris tuberosa L.	large canarygrass	--	NS
Phalaris tuberosa var. stenoptera (Hack.) Hitchc.	hardinggrass	NS	--
Phleum pratense L.	timothy	S	--
Poa ampla Merr.	big bluegrass	--	S
Poa annua L.	annual bluegrass	--	S
Poa compressa L.	Canada bluegrass	S	--
Poa pratensis L.	Kentucky bluegrass	S	S
Poa trivialis L.	roughstalk bluegrass	NS	S
Secale montanum Guss.	mountain rye	--	S
Setaria italica (L.) Beauv.	foxtail millet	S	--
Sorghastrum nutans (L.) Nash	Indiangrass	NS	S
Sorghum almum L.		S	S
Sorghum halepense (L.) Pers.	Johnsongrass	S	S
Sorghum sudanense (Piper) Stapf	Sudangrass	NS	--
Sorghum vulgare Pers.	sorghum	--	S
Sporobolus airoides (Torr.) Torr.	alkali sacaton	S	S
Sporobolus cryptandrus (Torr.) A. Gray	sand dropseed	S	--
Sporobolus giganteus Nash	giant dropseed	--	NS
Stipa cernua Stebbins & Love	nodding needlegrass	S	--
Stipa viridula Trin.	green needlegrass	S	S
Trichachne californica (Benth.) Chase	cottontop	--	S
Trisetum flavescens (L.) Beauv.	golden oatgrass	--	NS
Zoysia japonica Steud.	Japanese lawngrass	NS	--
	Meyer Zoysia	NS	--
Zoysia japonica Steud. x Z. tenuifolia Willd.	Emerald Zoysia	NS	--
Zoysia matrella (L.) Merr.	Manilagrass	NS	--
Zoysia tenuifolia Willd.	Mascarenegrass	NS	--

[a] S = susceptible, NS = non-susceptible, -- = not tested.

a variety of known susceptibility, was entered in each trial. Inoculations were accomplished 4 weeks from the time of seeding, with the inoculum being prepared in the following manner: Pythium ultimum, isolated from bentgrass, was grown in potato-dextrose broth for 12 days at 22° C, after which the mycelial masses were removed from the broth, washed in distilled water, cut into small units with shears, and suspended in distilled water. One hundred ml of the aqueous mycelial suspension was poured over the foliage of the plants of each container, and the plants were maintained in a moisture-saturated atmosphere in a greenhouse for 14 days at approximately 28° C. Uninoculated plants of each species studied served as checks.

At the end of the incubation period, the reaction of the various grass species to P. ultimum was recorded (Table 1). No attempt was made to assess relative degrees of susceptibility.

The parasitic capability of H. vagans was tested on 69 species of 33 genera of Gramineae. As in the Pythium blight investigation, the seed was sown in 6-inch plastic pots in a soil mixture. In each trial, to determine whether conditions were optimum for disease development, Kentucky bluegrass, a species of known susceptibility, was also tested. In addition, uninoculated plants of each species tested served as checks.

Inoculum was prepared by growing H. vagans, isolated from Kentucky bluegrass, on potato-dextrose agar slants for 5 weeks at 20° C. An aqueous suspension of mycelium and conidia was sprayed on the plants and then the plants were placed in a humid atmosphere in a greenhouse at approximately 21° for 10 days. Seventeen days after inoculation disease incidence based on leaf lesion formation was recorded.

In view of the possibility of seed-borne Helminthosporia, reisolation of the pathogen was attempted with all species exhibiting typical Helminthosporium lesions. Only those species from which H. vagans was reisolated are recorded as being susceptible (Table 1).

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, THE PENNSYLVANIA STATE UNIVERSITY, UNIVERSITY PARK, PENNSYLVANIA

PERFORMANCE OF CERTAIN NEMATOCIDES AND NEMATOCIDE-FERTILIZER MIXTURES APPLIED TO VEGETABLE CROPS IN NORTH CAROLINA

Robert Aycock and J. N. Sasser[1]

Abstract

DBCP-fertilizer mixtures were as effective in nematode control on certain vegetable crops as granular DBCP and fertilizer applied separately. In tests where nematode populations were moderately high, significant increases in yields resulted from use of certain nematocides and/or nematocide-fertilizer mixtures if the roots of the plants were not injured by fertilizer. When okra was seeded directly over a band of fertilizer or DBCP-fertilizer mixture, injury occurred, thus placement is an important factor. The application of DBCP to tomatoes at setting time depressed yields of tomatoes but pre-setting applications with a 10-day waiting period resulted in no phytotoxicity.

INTRODUCTION

Nematode diseases are the principal limiting factor in the production of vegetable crops in the intensively farmed Castle Hayne area of southeastern North Carolina. Principal parasitic nematodes involved are root-knot, Meloidogyne incognita acrita Chitwood 1949, and M. hapla Chitwood 1949; stubby root, Trichodorus christiei Allen; and spiral, Helicotylenchus nannus Steiner 1945 and Scutellonema sp. The availability of nematocides such as 1,2-dibromo-3-chloropropane (DBCP) which can be absorbed on clay granules and used in fertilizer mixes before or after planting has increased the interest of growers in soil fumigation because of simplicity of application resulting in reduced cost.

Although the nematocidal value of DBCP is well known (6), there is some disagreement in reports concerning phytotoxicity of this material (2, 3, 4, 5). Since little information is available on the performance in North Carolina of DBCP-fertilizer mixtures, investigations were conducted on the response of various vegetable crops including okra, snap beans, squash, tomato, and turnips to DBCP and other soil fumigants.

REVIEW OF LITERATURE

McBeth and Bergeson (6) in preliminary phytotoxicity experiments found onion and tobacco transplants as well as seedlings of tobacco, onion, lettuce, beets, and cole crops sensitive to DBCP. Intermediate in reaction were tomato seedlings and sweetpotato and tomato transplants. Numerous other fruit, vegetable and ornamental species were tolerant of DBCP. Lear and Thomason (4) obtained large increases in yield of tomatoes with row application of DBCP at 1 gallon/acre rate (5-foot rows). In subsequent tests where transplants were set 3 weeks after fumigation, broadcast applications of 1.5 and 2.5 gallons/acre and 0.6 gallon/acre row-placement depressed yields significantly. When dosage was decreased to 0.75 gallon/acre broadcast or 0.3 gallon/acre row-placement no depression of yields occurred but roots in all DBCP treated plots were brown, coarse textured and had few laterals.

Kantzes, et al. (3) used 25% granular DBCP in fertilizer mixtures on the eastern shore of Maryland at rates of 24 and 35 pounds/acre and obtained excellent control of root-knot nematodes on cantaloupe and tomatoes. Fertilizer-nematocide mixtures were applied to cantaloupes at planting time and to tomatoes 1 week after transplanting. Although DBCP was highly effective in root-knot, Meloidogyne incognita acrita, control, yields were not reported.

Good (2) obtained excellent control of root knot with a liquid formulation of DBCP at races of 0.25, 0.50, and 1.0 gallon/acre (38-inch rows) applied 9 days before planting. Yields of tomatoes and cowpeas, however, were decreased slightly with increased rates. Time of setting application and preplanting applications of DBCP were compared with 1,3-dichloropropene-1,2-dichloropropane (D-D) (10 gallons/acre) on tomatoes; significant increases in yield were noted only with D-D. Visual evidence of phytotoxicity to vines and root abnormalities were observed. Tomato yields from 0.5 gallon and 0.75 gallon/acre plots (4-foot rows) were significantly smaller than those from D-D treated plots, probably indicating toxicity.

[1]Research Associate Professors of Plant Pathology, North Carolina State College, Raleigh, North Carolina.

PROCEDURE

Evaluations were made during the period 1958 through 1960. Materials usually were applied as row treatments in small plots 25 to 50 feet long. Rows were 6 feet wide for tomatoes and 3 feet for other crops.

Furrows were opened with a small shovel plow and weighed or measured amounts of the fumigant were applied 5 to 6 inches deep. Ridging followed immediately with a turning plow. Soil moisture was near field capacity and soil temperatures were between 50° and 70° F at a depth of 6 inches.

Liquid or granular DBCP, or granular DBCP mixed with fertilizer was used. Formulations of DBCP (Nemagon)[2] were an emulsifiable concentrate (8.6 pounds DBCP/gallon) and 17.3% granular (1 gallon active DBCP absorbed in 100 pounds Attaclay). DBCP-fertilizer mixtures were obtained by agitating manually cardboard drums containing the appropriate proportions of granular Nemagon and fertilizer. In each experiment, mixtures were applied to the soil on the same day they were mixed.

RESULTS

Experiments with DBCP: In 1958 plantings of okra (Clemson Spine-less), snap beans (Wade), squash (Early Summer Yellow-Straightneck) and tomato (Valiant) were made in soil that had been treated with various rates of DBCP (Table 1). A moderate infestation of stubby root and spiral nematodes was present. Root-knot nematode infestation was low at planting time. Galls were barely detectable in control plots at harvest. In certain experiments, in-the-row treatments of sodium n-methyldithiocarbamate (Vapam) and dimethyl tetrahydrothiadiazine thione (Mylone) were also included. All treatments were applied 10 to 14 days prior to planting or setting.

Significant differences in yield and plant vigor occurred only in the okra experiment (Table 1). Vapam and the 1.0 gallon/acre rate of DBCP (granular) increased yields over all other treatments. No evidence of phytotoxicity was detected in any of the experiments.

DBCP granular at 0.5 gallon/acre did not significantly increase yields of Purple Top turnip over the control (Table 2). Significant increases were obtained with ethylene dibromide (Dowfume W-85), methyl bromide-trichloronitromethane (Dowfume MC-2) and Mylone. In a previous experiment DBCP at 0.75 and 1.0 gallon/acre gave significantly greater yields of turnips than the control, however, when the half-gallon rate did not.

Populations of Trichodorus shortly after fumigation varied from 0 to 80/pint of soil. At harvest 10 weeks later, however, increases in nematode populations had occurred in all plots except in those where Dowfume MC-2 and Vapam were used (Table 2).

Experiments with DBCP-Fertilizer Mixtures: In 1959 and 1960 the response of tomatoes, okra and squash to DBCP and DBCP-fertilizer mixtures was investigated. In addition to DBCP, the effects of pentachloronitrobenzene (Terraclor) and Vapam on yield and disease incidence were also studied.

Tomatoes (Rutgers variety) were grown on soil having a very low infestation of root-knot nematodes. When applied at setting time, DBCP appeared to depress yields (Table 3). The emulsifiable concentrate was apparently more phytotoxic than the granular formulation. The former resulted in a rather serious drop in production when applied as a sidedressing while the latter applied as a sidedressing resulted in slightly increased (although not significantly so) yields over the control.

When Rutgers tomato plants were set 11 days after soil was treated with DBCP, no significant differences in yield occurred and no phytotoxicity was indicated. The application of 25 pounds and 60 pounds of Terraclor per acre increased yields of marketable fruit somewhat because of the presence of Sclerotium rolfsii Sacc., but these differences were not significant.

Where fertilizer alone or fertilizer-DBCP mixtures were applied directly under the bed 15 days prior to seeding, marked differences in vigor of okra plants were noted early in the season when these treatments were compared with those in which the fertilizer was applied in two bands beneath, but to the side of, the center of the bed (Table 4). Apparently these early season differences in vigor were caused by fertilizer injury to the young plants. Although this injury was very noticeable, yields of plots in which fertilizer-DBCP mixtures were applied directly under the row did not differ significantly from plots in which the materials were applied separately (Table 4). At the 1.0 gallon/acre rate both methods of application significantly increased yields over that of nontreated plots.

[2]Appreciation is expressed to Mr. Gus Kirchoff and Shell Chemical Corporation for materials and assistance.

Table 1. Effect of preplant applications of soil fumigants on vigor and yield of okra and population of stubby root nematodes.

Fumigant	Rate/acre	Mean vigor[b]	Mean yield (pounds)	Average no. nematodes/pint of soil[c]
DBCP[a]	.5 gal.	1.7	2.6	182
DBCP	.75 gal.	2.5	3.2	147
DBCP	1.0 gal.	2.5	4.1	60
Mylone	.50 lb	2.5	3.0	93
Vapam	16 gal.	4.5	6.2	205
Control		2.2	2.6	75
LSD 5%		.8	1.1	-
1%		1.1	1.6	-

[a]Granular (17.3%)
[b]Vigor based on 1-5 scale with rating of 1 being least vigorous.
[c]Soil assay for nematodes made at mid-season.

Table 2. Effect of soil fumigation on nematode populations and yield of turnip.

Fumigant[a]	Rate/acre	Nematodes/pint of soil[b] stubby root	Nematodes/pint of soil[b] spiral	Yield (pounds)
Dowfume W-85	2 1/4 gal.	750	0	22.8
D-D	5 gal.	975	250	19.8
Vapam	16 gal.	67	15	15.6
Dowfume MC-2	2 lb./100 sq. ft.	0	0	29.7
DBCP	.5 gal. granular	525	0	20.8
Mylone, 50D	50 lb	900	15	22.3
Control		1475	375	13.2
LSD 5%		-	-	7.9

[a] All treatments were in-furrow applications on rows of 3-foot spacings except for methyl bromide which was applied as a solid treatment under cover.
[b] Soil samples for nematode assay were taken at harvest approximately 11 weeks after application of nematocides.

Table 3. Effect of in-the-row applications of DBCP and DBCP-fertilizer mixtures on yield of Rutgers tomatoes.

Rate/acre (gallons)	Formulation and method of application	Mean yield (no. marketable fruit)
0.25[a]	granular, 17.3%	53
0.25[a]	granular, 17.3% mixed in fertilizer	52
0.25[a]	50% emulsifiable	50
0.25[b]	granular, 17.3% side-dressing	66
0.25[b]	50% emulsifiable, side-dressing	33
Control[c]		61
LSD 5%		10
1%		16

[a] Applied at time of setting.
[b] Applied 6 weeks after setting as sidedressing in furrows opened about 6 inches from center of bed on either side. Rows 6 feet apart.
[c] Fertilizer only (5-10-10) applied in two bands below and at side of bed at rate of 1000 pounds/acre. DBCP and DBCP-fertilizer mixture was applied in furrow 5 to 6 inches deep and immediately ridged upon.

Soil samples from within each plot were composited and set to Rutgers tomato plants in the greenhouse. The number of galls that developed in 40 days are shown in Table 4. Although differences in degree of control are indicated between the 0.5 and 1.0 gallon/acre rate, there was no apparent advantage in applying nematocide and fertilizer separately.

In a similar experiment with Early Yellow Summer Squash, yield and control data were comparable to that obtained with okra, except that no injury was noted when fertilizer placement was in a single band under the row. Significant increases in yield resulted from DBCP fumigation but applying DBCP separately or in fertilizer mixtures did not affect fumigation results as reflected in yield or nematode control. The 1.0 gallon/acre rate gave a high degree of root-knot control as indicated by gall counts on plants.

Table 4. Effect of preplanting applications of DBCP and DBCP-fertilizer mixtures on yield and root-knot nematode control in Clemson Spineless okra.

Treatment	0.5 gallon/acre rate[a]		1.0 gallon/acre rate	
	no. galls[e]	yield	no. galls	yield
DBCP-fertilizer mixture[b]	13.6	14.4	1.0	17.2
DBCP-fertilizer, applied separately[c]	38.2	16.4	0.5	14.8
DBCP only[d]	1.25	9.8	1.0	9.7
Control, fertilizer only[d]	59.0	11.6	111.5	8.6
LSD 5%	--	3.2	--	3.2
1%	--	4.5	--	4.5

[a] Based on row spacings of 3 feet.
[b] Fertilizer (5-10-10) mixed with 17.3% granular DBCP applied at 600 pounds/acre in single furrows 5 to 6 inches deep and immediately ridged upon.
[c] DBCP applied in furrow 5 to 6 inches deep and immediately ridged upon. Fertilizer applied in two bands below and at each side of bed.
[d] Applied in single furrows 5 to 6 inches deep and immediately ridged upon.
[e] Galls formed in 40 days on Rutgers tomato plants grown in soil samples obtained from each plot at mid season.

DISCUSSION

These data confirm previous reports (2, 4) that tomatoes are not as tolerant of DBCP as certain other plants. Both granular and liquid formulations applied at setting time resulted in depressed yields. Although the liquid formulation applied as a sidedressing 6 weeks after setting had severe phytotoxic effects, the granular formulation did not. Where treatments were applied 10 days prior to setting, no depression in yield occurred. These findings emphasize the need for caution and judgment in recommendations involving the use of DBCP and indicate that a waiting period between fumigation and setting of tomato transplants is desirable in southeastern North Carolina. No increase in the incidence of S. rolfsii was noted as reported by Rankin and Good (8) on soil fumigated with nematocides.

Results from these experiments show, however, that DBCP-fertilizer mixtures generally performed as well as granular formulations of DBCP applied separately. No differences in nematode control between the two methods of application were detectable. Fertilizer injury occurred, however, when okra was seeded directly over a band of fertilizer or DBCP-fertilizer mixture. Thus fertilizer placement must be considered if DBCP-fertilizer mixtures are to be used. Plants with tap roots are probably more likely to be injured when fertilizers or fertilizer-nematocide combinations are placed directly under the center of the bed. It was postulated that high yields of okra in Vapam treated plots resulted from control of other soil organisms in addition to nematodes.

Rather high populations of spiral and stubby root nematodes are often recovered from soils in which turnips are growing in North Carolina. In fumigation experiments reported here, populations of Trichodorus increased considerably in all plots except where Dowfume MC-2 and Vapam were used (Table 2). In Florida (7) build-up of this species following fumigation continued until the population was 3 to 4 times that of comparable unfumigated plots. Excellent kills were reported with D-D, EDB, and MC-2 under a cover. Row treatments, however, were ineffective. Christie (1) suggested that materials like DBCP which tend to linger in the soil might retard the rapid buildup of Trichodorus, but such did not occur in experiments discussed here (Table 2).

It should be emphasized that these studies do not take into account the possible changes that might ensue in DBCP-fertilizer mixtures stored for considerable periods prior to usage. Neither have the possible effects of DBCP upon changes of fertilizer in the soil nor the possible effects of different sources of N, P_2O_5 and K_2O upon the DBCP and vice versa been investigated. It is therefore not implied that these data are sufficient to recommend the inclusion of DBCP with fertilizers. However, DBCP (Nemagon) was as effective when mixed with fertilizers as when applied separately under the conditions of these tests.

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DEPARTMENT OF PLANT PATHOLOGY, NORTH CAROLINA STATE COLLEGE, RALEIGH AND HORTICULTURAL CROPS RESEARCH STATION, CASTLE HAYNE, NORTH CAROLINA

IRISH POTATO SEED PIECE TREATMENT WITH VARIOUS CHEMICALS

J. B. Sinclair[1]

Abstract

Seed pieces of Red LaSoda variety of potatoes were dusted or dipped in various concentrations of potassium gibberellate (0-100 ppm) or in .83% Agri-mycin-500 (1.76% streptomycin, 42.40% basic copper sulfate and 0.18% oxytetracycline), 5% 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene), 5% and 10% each pentachloronitrobenzene (PCNB) plus captan, 6.5% zineb and 7.6% ziram. Potassium gibberallate and streptomycin were generally found to be phytotoxic at the concentrations used, and reduced stands and yields significantly below the nontreated checks. PCNB plus captan and zineb reduced stands and yields below the checks, but the differences were not significant.

INTRODUCTION

The Irish potato industry in Louisiana is comparatively small, but important to the people of the area where potatoes are grown. In 1960 Louisiana produced 784,000 bushels of Irish potatoes with a market value of approximately 1 1/4 million dollars (9). Irish potatoes are usually planted in Louisiana during January or early February. Under the cold, wet conditions prevalent during this period, germination may be slow and uneven, and seed piece decay may cause a certain amount of reduction in stand. A series of field tests were planned to test whether the use of potassium gibberellate, as a seed piece treatment, might give rapid germination and uniform stand and whether selected fungicides would control seed piece rot.

MATERIALS AND METHODS

Certified Irish potatoes of the variety Red LaSoda and/or Triumph were used in all tests. Tubers were cut, treated and planted the same day to coincide with commercial plantings. Uniform seed pieces were either dusted with or dipped in solutions of potassium gibberellate ranging in concentrations from 0 to 100 ppm (Table 1). All fungicides were applied as dusts and included: .83% Agri-mycin-500 (1.76% streptomycin, 42.40% basic copper sulfate, and 0.18% oxytetracycline), 5% 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene), 5% and 10% each of pentachloronitrobenzene (PCNB) plus captan, 6.5% zineb and 7.6% ziram (Table 2). Nontreated seed pieces served as controls in both cases. The tests were made for 4 years in randomized, replicated field plots. The seed pieces were planted 1 foot apart. Seed pieces per plot for the potassium gibberellate dust series numbered 30 in 1958 and 1959 and 50 in 1960. There were 40 seed pieces per plot used in the liquid potassium gibberellate dip test in 1959. The fungicide series had 40 seed pieces per plot in 1959 and 1958, and 50 in 1959 and 1960. Stand counts, yield of marketable and cull tubers were taken and adjusted to percentage stand and yields in in bushels of marketable tubers (Tables 1 and 2). The plots were located in the same general vicinity for 4 years.

RESULTS

Data from results using the variety Red LaSoda are given in Tables 1 and 2. Practically identical data were obtained from seed pieces of the Triumph variety. Portions of these data have appeared in abstract form (5, 6, 7, 8).

Potassium gibberellate, when used as a dust, neither increased stands nor gave a uniform stand in three trials at the concentrations used (Table 1). In 1960 the stands were significantly below that of the nontreated check. Seed pieces dipped in a solution of 5 and 10 ppm potassium gibberellate in 1959 showed an increase in stand above the check, but the difference was not significant. At 25 ppm, the stand was the same as the check. In all cases, yields were significantly below that of the checks, but there were no significant differences in cull tubers produced.

None of the materials used in the seed piece rot control studies controlled the disease, which caused at least a 10% reduction in stand as indicated by the nontreated checks. Agri-mycin-500 at the rate used was phytotoxic, as shown by stand and yield reductions significantly

[1]Associate Professor of Plant Pathology, Louisiana State University Agricultural Experiment Station, Baton Rouge.

Table 1. Treatment means for year of test, percentage stand and yield of marketable tubers from seed pieces either dusted or dipped in concentrations of potassium gibberellate indicated.

Treatment	Rate (ppm)	Percentage Stand				Yield (bushels/acre)			
		1958	1959	1959	1960	1958	1959	1959	1960
Dust	0	87	90		90	264	83		150
	5		80		80		34		25
	10		80		78		28		51
	25	73	87		80	264	22		21
	50	87				239			
	100	90				195			
Dip	0			87				61	
	5			93				32	
	10			96				27	
	25			87				16	
LSD 05		ns	ns	ns	4	22	22	7	30
LSD 01		ns	ns	ns	ns	29	ns	14	43

Table 2. Treatment means for year of test, percentage stand and yield of marketable tubers from seed pieces with certain fungicides with percentage of active ingredients indicated.

Treatment	% active ingredients	% stand				Bu/A Yield			
		1957	1958	1959	1960	'58	'59	'59	'60
Agri-mycin-500	.83		85	66	56		140	81	98
Dyrene	5		92				243		
PCNB +	5 ea.			96				120	
captan	10 ea.				82				163
Streptomycin nitrate	.01	90				372			
Zineb	6.5	90	90	98		361	237	120	
Ziram	7.6				90				176
Check		85	90	98	86	389	237	141	154
LSD 05		3	3	4	8	ns	11	ns	17
LSD 01		5	5	6	12	ns	16	ns	26

below those of nontreated checks (Table 2). Other materials that showed stand and/or yield reductions below the check were: PCNB + captan, streptomycin nitrate and zineb, although these differences were not significant. Dyrene-treated seed pieces gave a stand significantly above the check, but the same relationship was not found in yield. There were no significant differences in the number of cull tubers produced.

DISCUSSION

Results from tests using streptomycin nitrate are not in complete agreement with Bonde (1), who reported a significant increase in stand and yields above the nontreated checks from seed pieces dipped in 50 and 100 ppm streptomycin nitrate. Results reported in this paper show that .01% (100 ppm) streptomycin nitrate gave a significant increase in stand above the check, but was followed by a yield reduction. A single test may not be enough to draw final conclusions; however, a 3-year experiment by Fink (2) showed that streptomycin sulfate reduced stands and yields significantly below nontreated checks. Logsdon (3) and Ruehle (4) have reported similar results.

A concentration of 1 ppm of potassium gibberellate was reported to give early and uniform emergence (4). The rates used in the experiments reported in this paper may have been too high to be effective.

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LOUISIANA STATE UNIVERSITY, BATON ROUGE, LOUISIANA

COMPARISON OF STANDARD FUNGICIDES RECOMMENDED FOR CONTROL OF POWDERY MILDEW OF ROSES[1]

Ira W. Deep and Allan Bartlett[2]

Abstract

Eight treatments for control of powdery mildew of roses were compared in plots where blackspot was not severe. Treatments with Karathane, Acti-dione PM, Phaltan spray, and copper oleate (Mildew King) were about equally effective and all were more effective than the sulfur spray, sulfur dust and Phaltan dust treatments. An Indopol Polybutene treatment appeared to have eradicative action but was phytotoxic. All the fungicides except Karathane effectively controlled black spot where this disease was not severe.

INTRODUCTION

The two most important diseases of roses in the United States are powdery mildew, caused by Sphaerotheca pannosa (Wallr.) Lév., and blackspot, caused by Diplocarpon rosae Wolf. Both are widespread but in the East and South blackspot is more prevalent than mildew, while in the Pacific Northwest blackspot is relatively less important and powdery mildew is especially severe. Investigators setting up spray trials in the East to determine the effectiveness of fungicides in controlling mildew have had to cope with blackspot, and various spray combinations designed to control both diseases have usually been tested (4, 6). Mildew trials have recently been run on blackspot-resistant Rosa wichuraiana (7). The severe mildew and relatively less severe blackspot in the Pacific Northwest make this area particularly suited for tests of rose mildew sprays.

MATERIALS AND METHODS

The experimental plot consisted of 360 plants, 120 plants each of the three susceptible varieties: Nocturne, White Knight, and Poinsettia. The plants were arranged in 60 blocks with each block containing six plants, two of each variety. The six-plant block was used as a single sample in collecting data. The plot was divided into five replicated units with 12 blocks in each unit, and the eight treatments and two controls were randomized within each unit. Each spray material was applied at the dosage ordinarily recommended for control of powdery mildew of roses. Triton B-1956 was added to all materials applied in spray form. Materials tested were 2-(1-methylheptyl)-4,6-dinitrophenyl crotonate (Karathane), cycloheximide (Acti-dione PM), N-trichloromethyl thiophthalimide (Phaltan) spray, Phaltan dust, sulfur spray, sulfur dust, Indopol Polybutene, and copper oleate (Mildew King).

Treatments were made each Friday from August 12th through October 7th. Data were taken each Thursday from September 8th through October 20th. A disease rating system of 0 to 11 was employed with 0 equal to no mildew and 11 equal to very severe mildew. Data were always taken by two individuals working independently, and the data in Figure 1 represent the average of the readings of both individuals.

The incidence of blackspot was very low until the first week in October when it developed fairly rapidly. Control plants were moderately heavily infected by October 16th when data were taken. In collecting blackspot data the degree of infection of each individual plant was considered rather than taking the six-plant block as a sample. Although all the plants were quite susceptible to mildew, the variety Poinsettia was definitely resistant to blackspot and White Knight appeared to be somewhat less susceptible than Nocturne.

RESULTS

Comparative Effectiveness in Control of Powdery Mildew: There was very little difference in the effectiveness of Karathane, Acti-dione PM, Phaltan spray, and Mildew King in controlling powdery mildew (Fig. 1). Phaltan dust and sulfur spray ranked next, and sulfur dust was least effective of these seven treatments. All of the treatments markedly reduced mildew as compared with the control. In many cases the rose plants with slightly more mildew had a more desirable appearance because some of the more effective treatments were mildly phytotoxic or left chemical residues.

[1]Technical Paper No. 1440, Oregon Agricultural Experiment Station. This investigation supported in part by a grant from the American Rose Foundation.

[2]Assistant Professor of Botany and Research Assistant, respectively, Oregon State University.

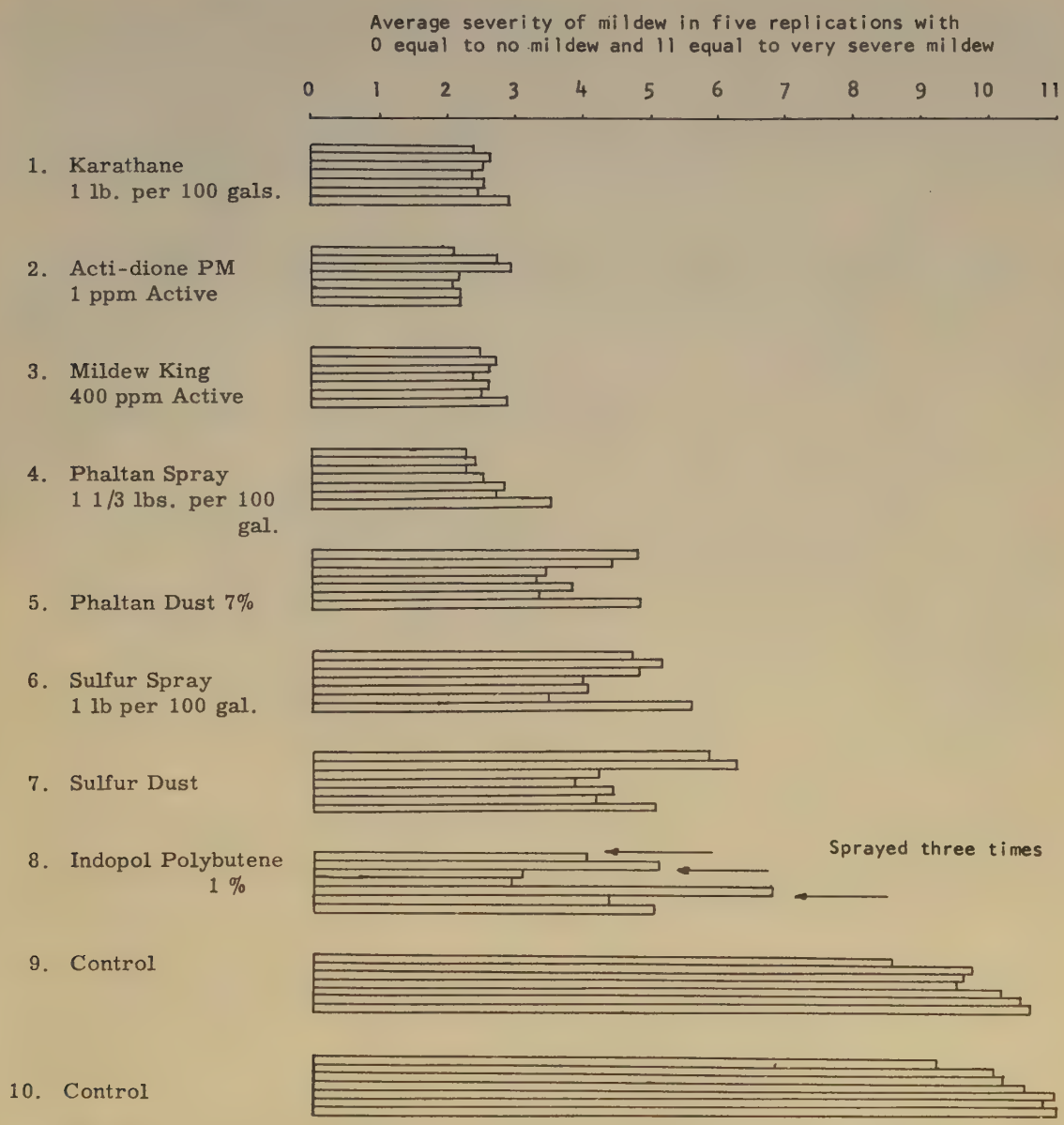
Average severity of mildew in five replications with
0 equal to no mildew and 11 equal to very severe mildew
0 1 2 3 4 5 6 7 8 9 10 11
1. Karathane
1 lb. per 100 gals.
2. Acti-dione PM
1 ppm Active
3. Mildew King
400 ppm Active
4. Phaltan Spray
1 1/3 lbs. per 100
gal.
5. Phaltan Dust 7%
6. Sulfur Spray
1 lb per 100 gal.
7. Sulfur Dust
8. Indopol Polybutene
1 %
Sprayed three times
9. Control
10. Control

FIGURE 1. Severity of rose powdery mildew each week for seven weeks with treatments applied once a week six days prior to taking data.

Acti-dione PM was slightly more effective than the other materials in controlling mildew toward the end of the season, but the leaves on plants that had been treated with this material at weekly intervals for several weeks were dwarfed. Some of the leaflets were slightly distorted and there appeared to be an unusually large amount of anthocyanin in the young leaves. There was no necrosis and the plants were not stunted. Less frequent application of this material might have controlled mildew without injury to the plants. In similar trials conducted in 1959 Acti-dione PM gave the most complete control of mildew and caused no noticeable injury (1).

The Phaltan spray and dust treatments and the sulfur dust treatment left residues. Phaltan spray was very effective in controlling mildew while Phaltan dust and sulfur dust were less effective. In all three cases, however, the plants were very vigorous and the leaves were well expanded and healthy in appearance.

Karathane and Mildew King were quite effective, caused no noticeable injury, and left no appreciable residue under the conditions of this experiment. Karathane has been reported to cause injury when applied during periods of high temperature. The maximum temperature was seldom above 85° F during the course of these trials.

Indopol Polybutene was applied on only three dates, September 2nd and 16th and October 7th. Following the treatment on September 16th, there was a marked reduction in mildew for 2 weeks and the rose leaves had an attractive sheen, but by the third week mildew had increased sharply. The October 7th treatment again brought about a marked reduction in mildew. This material appeared promising, but after 3 weeks a phytotoxic reaction developed. The treated leaves of all three varieties turned yellow and eventually died. Polybutene tested by other investigators on the rose variety Pink Garnet Supreme was reported to give excellent control and no phytotoxicity (2, 3).

Effectiveness in Control of Blackspot: Blackspot developed only during the last 2 weeks of these trials and was never very severe. All of the treatments except Karathane gave effective control of blackspot under these conditions (Table 1). Phaltan spray was the only treatment that completely prevented development of blackspot, but there was little infection following treatment with Mildew King or sulfur.

Table 1. Relative effectiveness in control of blackspot. Disease ratings are based on a 0 (no disease) to 11 (severe disease) rating system.

Treatment	Average severity of blackspot on 10 plants of rose variety:		
	White Knight	Nocturne	Poinsettia
1 Karathane	2.3	4.0	0.5
2 Acti-dione PM	0.6	0.4	0.0
3 Mildew King	0.4	0.0	0.0
4 Phaltan spray	0.0	0.0	0.0
5 Phaltan dust	1.5	1.5	0.0
6 Sulfur spray	0.6	0.0	0.0
7 Sulfur dust	1.2	2.1	0.3
8 Indopol Polybutene	0.8	0.2	0.0
9 Control	4.4	5.2	1.3
10 Control	4.9	5.4	2.9

Phaltan and sulfur dusts were less effective in controlling black spot than were the spray treatments with the same materials.

DISCUSSION

Four of the materials now used extensively for powdery mildew control were about equally effective under environmental conditions especially conducive to severe mildew development on rose: Karathane, Acti-dione PM, Mildew King, and Phaltan in spray form. Three other treatments resulted in less thorough but still acceptable control of mildew: sulfur spray, sulfur dust, and Phaltan dust. Indopol Polybutene was rather effective and gave some eradicative action but was phytotoxic. Acti-dione PM was mildly phytotoxic, causing dwarfing of the leaves.

There appeared to be cycles of increase and decrease in mildew during the trials. The cycles were not due to suppression of disease by the fungicides alone since there was a consistent increase or decrease in mildew each week regardless of the treatment. This suggested the influence of weather conditions on mildew severity, but the cycles did not correlate well with the average temperature, relative humidity, and rainfall information available. For example, in the week prior to September 14th the average maximum temperature was 84° F and the average relative humidity at 8:00 a.m. was 69%. According to spore germination tests made by Longree (5) these conditions might be expected to suppress mildew, yet there was an increase in severity of mildew. During the following 2-week period the temperature was lower, averaging 73° and 76°; the relative humidity was higher, averaging 85% and 90% at 8:00 a.m., and there was no rain. Mildew could be expected to develop profusely under these conditions, but there was a decrease in mildew each week.

The decrease in mildew on October 12th is probably correlated with rainfall during the previous week. Rain fell on 5 of the 7 days, and it has been shown by Yarwood (8) that a water spray alone may considerably reduce the severity of powdery mildew of rose.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY,
OREGON STATE UNIVERSITY, CORVALLIS, OREGON

WHEAT RUST DURING THE SPANISH ERA IN CALIFORNIA

Ralph E. Smith[1] and Jacob N. Bowman

With respect to plant diseases in the Spanish era in California, most of the priests and explorers who left behind their mission records, diaries, and descriptions of their travels apparently recognized only one. This was wheat rust, or chauistle as it was called. This word was written in various ways, such as chauistle, chahuistle, chaguiscle, chaquistle, chauiste, chaguiste, chaguistle and chahuiztle. Rolulu lists it as of Aztec origin and writes it chaguiscle. The fact that the Aztecs had a name for it lends weight to the idea that wheat rust was endemic in Mexico or introduced at a very early date.

Simpson (7, p. 142), referring to Mexico, relates that in the year 1691-1692, "...unseasonable and excessive humidity brought on a blight of mildew (chahuixtle) and killed the whole wheat crop." Humboldt (4, p. 436) in 1803-04 records that in Mexico, "The rye and barley are seldom attacked by a disease called by the Mexicans, chaquistle, which frequently destroys the finest wheat harvests...". Baegert (1, p. 23), who was in Lower California from 1751 to 1768, states that, "Sometimes the fogs carry with them something the Mexicans call chahuixtle, which is very damaging to the wheat fields."

Referring to Mission Purisima de Cadegomo in Lower California in 1772, Palou (6, p. 192) says, "At present they have seven bushels of wheat sown, and if it escapes the chahuistle they will harvest a fine crop." Referring to Mission San Francisco Xavier in Lower California, he says (6, pp. 182-183), "It has at present a field of corn which promises well, and a good crop is hoped for if God saves it from the chauistle, which is apt to ruin crops at the best time." He thus applies the same term to wheat rust and to a disease of corn. In 1786, Clavigero (2, p. 45), discussing the growing of wheat in Lower California, says of one type of wheat "...little of it is sown in California, since it is much more subject to the blight known there as well as in Mexico by the Mexican name of chahuiztle."

The following manuscript references to wheat rust or chauistle in what is now the State of California have been found in the Bancroft Library and in the mission records in San Francisco, Fresno, Los Angeles, San Gabriel, and Santa Barbara:

San Gabriel report, in Patentes in Mission San Gabriel. 1778. The wheat harvest was only 55 fanegas because of the chaguistle.

California Archives (Bancroft Library) 23-III. 131. Report of Governor Fages. 1783. The harvest of wheat has been very expensive, due principally to the chahuistle.

California Archives, 17-XLII. 5. Missions, 1814. Some of the missions have lost their harvest of wheat through the chahuiste.

Archivo del Arzobispado (Bancroft Library), III. 5. Santa Cruz. 1817. The whole wheat harvest was lost, due to chauistle, but the frijol were not affected.

California Archives, 25-IX. 189. Southern California, 1818. From San Diego to San Luis Obispo among the pestilences was the chaguiscle which follow the continuous fogs.

Archivo de Santa Barbara, III. 275. Santa Ines, 1822. The pest which strikes wheat is the chaguiste.

Archivo de Santa Barbara, IX. 116. San Carlos, 1822. If the chauistle does not destroy the harvest, sowings will be made of all the seed...

Archivo de Santa Barbara, IX. 131; III. 278. Santa Barbara, 1822. The most terrible enemies which strike the sowings are the chauiste and the grasshoppers.

Archivo de Santa Barbara, III. 263. San Diego, 1822. This season they were not bothered by the chaguiste or drouth.

Hartnell, W. E. P., Diario (Bancroft Library), 187. 1839. San Fernando. This year at the mission they were not

[1] Professor Ralph E. Smith, who died in 1953, left among his manuscripts this information on wheat rust which is based to a considerable extent upon a manuscript by the historian Dr. Jacob N. Bowman, entitled "Notes on Rust (Chauistle) in Provincial California, "1943. This is in the Bancroft Library, University of California. M. W. Gardner.

bothered with grasshoppers or chahuistle.

In his Memoirs of California, 1829, Khlebnikov (5, p. 320), discussing wheat harvests, states, "In 1826 at the mission of San Luis Rey altogether one thousand fanegas were sown and only seven hundred were harvested. During the same year all seeds were injured by rust."

The teliospores and urediospores of stem rust were found by Hendry and Hansen (3) on wheat straw in adobe bricks from the Soledad Mission in the Salinas Valley. These date back at least to 1832, when the mission was rebuilt.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

BORON DEFICIENCY EVIDENT IN OREGON HOLLY ORCHARDS[1]

A. N. Roberts, R. L. Ticknor[2] and O. C. Compton

Abstract

Greenhouse sand cultures were used to develop boron deficiency symptoms in two commercial clones of Ilex aquifolium. Irregularly-shaped, reddish or purplish spots developed on the upper surface of mid-terminal leaves which on the lower surface were water-soaked in appearance. These spots progressively enlarged, developing a pattern of concentric rings bordered on the outside with yellow. In some instances the veins were enlarged in these areas. This injury often resulted in considerable defoliation. Although less frequent than spotting, a pitting of the upper leaf surface apparently resulted from buckling or collapse of the mesophyll. Leaves appearing near the end of a growth flush were sometimes misshapen and malformed. Boron deficiency symptoms were present under field conditions in some parts of Oregon. The deficiency level for mid-terminal leaves at the end of a growth flush was estimated as 15 to 20 ppm boron.

English holly, Ilex aquifolium, is a major item in the "greenery" industry of the Pacific Northwest. Market demands are for well-fruited sprays with dark green foliage free of blemishes caused by insects, diseases or faulty nutrition. Insect and disease problems of English holly have been summarized by Swenson (2) and Young and Buddenhagen (3), respectively. Buddenhagen and Young (1) found that Phytophthora ilicis Buddenhagen & Young would cause black leaf-spotting, subsequent defoliation and twig cankers. Several other forms of necroses have been described, many of which were of unknown origin (3). One of these disorders, a red or purple-red leaf spotting, has been found to be one of the symptoms of boron deficiency. This report presents the evidence in support of this diagnosis.

DEFICIENCY SYMPTOMS

Greenhouse sand cultures with modified Hoagland's nutrient solutions were used to develop boron deficiency symptoms in two commercial cultivars of I. aquifolium, Rederly and Curly.

Rederly cuttings, rooted in January 1959 and grown in cultures without boron from April 1, developed symptoms of deficiency early the following August. Similarly treated cuttings of the variety Curly developed symptoms somewhat more slowly. The symptoms (Fig. 1) first appeared on mid-terminal leaves as irregularly-shaped, reddish or purplish spots on the upper surface which on the lower surface were water-soaked in appearance. As the leaves aged, these spots progressively enlarged developing a pattern of concentric rings bordered on the outside with yellow. Sometimes an enlargement of the veins and finally defoliation accompanied this spotting. Although less frequent than spotting, a pitting of the upper leaf surface (Fig. 2) apparently resulted from buckling or collapse of the mesophyll. Leaves appearing near the end of a growth flush were often misshapen and malformed.

DEFICIENCY LEVEL

Mid-terminal leaves of the Rederly variety taken for leaf analysis at the first sign of boron deficiency from complete and minus-boron cultures contained 39.6 and 10.6 ppm (dry basis) boron, respectively. Similar samples taken from the variety Curly contained 25.3 and 6.1 ppm boron for complete and minus-boron cultures. Leaf samples taken from the Rederly plants in April of the second year before new growth started contained 26.4 and 15.1 ppm boron for complete and minus-boron cultures. Deficiency symptoms were present on leaves of the latter culture. Samples of mid-terminal leaves taken from these plants in July after completion of new growth contained 36.1 and 6.9 ppm boron for complete and minus-boron cultures. Leaves produced on the latter solution were showing signs of boron deficiency.

FIELD SURVEY

A survey conducted in 1960 and 1961 on the status of nutrition in Oregon holly orchards af-

[1]Technical Paper No. 1422, Oregon Agricultural Experiment Station.

[2]Located at North Willamette Branch Experiment Station, Aurora, Oregon.

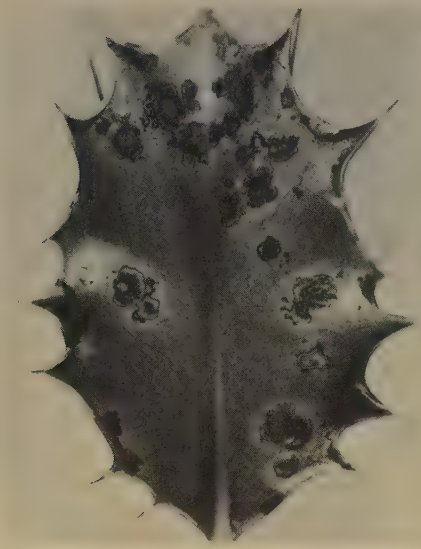

FIGURE 1. Leaf-spotting on English holly leaves resulting from boron deficiency.

FIGURE 2. Leaf-pitting on boron-deficient English holly leaves resulting from mesophyll buckling and collapse.

Table 1. Leaf boron content of English holly varieties in commercial orchards as related to boron deficiency symptoms (July sampling).

Variety	Boron deficiency symptoms in leaves: Present: orchard number	Present: boron (ppm)	Absent: orchard number	Absent: boron (ppm)
Rederly	70	11.9	2	21.3
	19	16.5	38	23.2
	4	20.8	56	33.1
	24	22.7	18	37.8
French-English	66	13.7	3	24.8
Green-stem (types)	72	11.0	13	28.5
	40	15.6	27	33.9
	41	16.5	32	44.3
	71	23.2	65	45.7

forded the opportunity of examing numerous commercial plantings for symptoms of boron deficiency and of estimating leaf deficiency level. Some of these data are given in Table 1.

These data indicate that incipient boron deficiency occurred in numerous commercial holly orchards in Oregon. The greenhouse trial and field survey showed that leaves containing 25 ppm boron or more were free of boron deficiency symptoms but that symptoms were always present when leaf boron was 20 ppm or less. Leaf boron levels of 21 to 24 ppm were found on both normal and deficient trees.

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DEPARTMENT OF HORTICULTURE, OREGON AGRICULTUAL EXPERIMENT STATION, CORVALLIS, OREGON

2,6-DICHLORO-4-NITROANILINE EFFECTIVE AGAINST RHIZOPUS FRUIT ROT OF SWEET CHERRIES

Joseph M. Ogawa, Stuart D. Lyda, and Darrell J. Weber[1]

Summary

A proprietary formulation (U-2069) of 2,6-dichloro-4-nitroaniline, used as a dip, was extremely effective in preventing Rhizopus rot (Rhizopus stolonifer) of the fruits of sweet cherry. At 2000 ppm the chemical was fungistatic, suppressing mycelial development in fruit tissues and preventing aerial mycelium from forming on the surface of infected fruit. It did not penetrate the uninjured epidermis in amounts sufficient to be detected, nor did it affect fruit respiration. A volatile material given off by the chemical was highly inhibitory to formation of aerial mycelium.

INTRODUCTION

The most common postharvest disease of stone fruits is caused by Rhizopus stolonifer (Fr.) Lind. Reduction of Rhizopus rot on stone fruits with chemicals has been reported (1, 2, 4), but no control is consistent. Packaged fruits of sweet cherry (Prunus avium) are quickly rotted by R. stolonifer. Initial infection occurs through breaks in the epidermis. The fungus later spreads to other fruit by aerial mycelium produced on the surface of these fruits (nesting effect). The disease could therefore be controlled by stopping mycelial development in the flesh of the fruit, or by preventing the stolons from establishing new infections on healthy fruit. Study of a chemical formulation (U-2069) capable of producing both effects is reported here. No data have been published on the performance of this chemical. The results of 1960 fungicide-nematocide tests (3) compare AL-50, which is an earlier name for U-2069, with other fungicides against Rhizopus rot of peaches, but the author did not report its formulation or source.

MATERIALS AND METHODS

Spores for inoculation were obtained by growing an isolate of R. stolonifer from peach (Prunus persica) on 1.5% potato-dextrose agar (PDA). To provide uniform infection of sweet cherry, a shallow slice was taken from the cheek of each fruit. A drop of water containing the spores was applied to the cut surface either before or after chemical treatment. The 2,6-dichloro-4-nitroaniline used was a 50% formulation (U-2069) supplied by the Upjohn Company, Kalamazoo, Michigan. This material is not yet approved for use on sweet cherries. The fruits were incubated in plastic containers with loose lids. Individual fruits were separated from each other on a wire mesh above a shallow layer of water in the container. All incubations were at 25° C. Uninoculated wounded fruits were used in each test. These uniformly failed to develop Rhizopus rot or brown rot caused by Sclerotinia fructicola (Wint.) Rehm or S. laxa Ader. & Ruhl.

RESULTS

Prevention of Fruit Rot: To test the preventive action of U-2069, 20 fruits each of Bing and Royal Ann cherry cultivars were wounded, dipped in a Rhizopus spore suspension, incubated 6 hours, and dipped in suspensions containing 500, 1000, 1500, and 2000 ppm of U-2069 (Fig. 1). At 36 hours all untreated fruit were rotted; no rot occurred in fruit treated with 1500 and 2000 ppm of the chemical; 80 and 25% of the fruit were partially rotted at 500 and 1000 ppm, respectively. In a second trial, Bing cherry fruits were handled as before except they were treated immediately with 5 to 2000 ppm of U-2069. Table 1 shows a slight reduction in rot at 5 ppm, and a gradual increase in effectiveness with increase in concentration. Rot was prevented for 30 hours at 1000 ppm, and for 72 hours at 1500 ppm, the 2000-ppm treatment did not give results significantly different from those of 1500-ppm treatment. In 168 hours, however, the percentage rot of fruit was 100 at 500, 32 at 1000, 4 at 1500, and 13 at 2000 ppm.

That the protection by U-2069 is fungistatic was shown as follows: fruits that were inoculated and treated but developed no rot were sectioned and examined under a microscope. Rhizopus mycelium was observed. Ten tissue transfers on PDA slants from these same areas resulted in growth.

[1] The authors thank Kenneth S. Anderson for taking the photographs, and the Upjohn Company, Kalamazoo, Michigan, for grant-in-aid for this project.

FIGURE 1. Preventive action against Rhizopus stolonifer through U-2069 dip treatments of 1000 ppm (B) and 2000 ppm (C) as compared with the untreated (A) after 43 hours of incubation at 25° C.

FIGURE 2. Suppression of Rhizopus rot with 2000 ppm dip of U-2069, 43 hours after inoculation and incubation at 25° C. (A) No treatment, (B) dipped 9 hours, (C) 18 hours, and (D) 30 hours after inoculation.

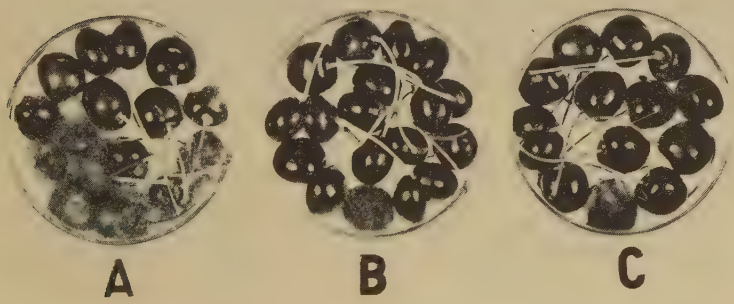

FIGURE 3. Nesting inhibited by U-2069 treatment of fruits in 6 days at 25° C. Extensive nesting onto untreated fruit (A) as compared with the slight (B) to no (C) movement of mycelium onto treated fruit.

Table 1. Percentage fruit rot on injured and inoculated Bing cherries treated by dipping in various concentrations of U-2069 and incubated at 25° C.

U-2069 (ppm)	Incubation period of fruits (hours)			
	30	43	72	168
0	40.8	100	--	--
5	34.0	100	--	--
10	31.0	100	--	--
50	25.5	100	--	--
100	13.3	66.3	100	--
500	0.5	11.0	32.5	100
1000	0	1.0	6.3	32.5
1500	0	0	0	4.3
2000	0	0	0.5	13.0

No chemical penetration of uninjured epidermis was detected. Forty uninjured Bing cherry fruits were dipped 3 minutes in 2000 ppm U-2069 and stored 20 hours at 8° C. These fruits were washed twice in 3 liters of distilled water, sliced to remove the epidermis and some mesocarp tissue, inoculated with Rhizopus spore suspension, and incubated. The rate of rot development and aerial mycelial growth did not differ from that of the untreated lot.

Respiration of Treated Tissue: The effects of the various concentrations of U-2069 on respiration of tissue of Bing cherry fruit were measured in a Warburg respirometer. Mesocarp slices or epidermal strippings were suspended in 3 ml of 0.1M phosphate buffer, pH 6.5. One ml of 500, 1000, and 2000 ppm U-2069 was introduced from the side arm after 1/2 hour of equilibration at 25° C. Plotting the μl of oxygen uptake per 100 mg dry weight of tissue at 30-minute intervals for 210 minutes showed no significant differences between the controls and the treated slices and strippings.

Suppression of Mycelial Development of Infected Fruit: The suppression of mycelial development by 200 ppm U-2069 dip treatment was tested. Forty Bing cherry fruits were used for each treatment. Injured fruits inoculated with spore suspensions were dipped after 9, 18, and 30 hours of incubation. No rot developed on fruits dipped 9 hours after inoculation (Fig 2B), when average length of germ tube in the flesh was 136.5μ. In 18 hours the average depth of mycelial penetration was 1.5 mm; treatment at this time permitted only internal rot development in the next 12 hours (Fig. 2C), but 20.5 and 72.5% external rot developed in 26 and 47 hours, respectively. In 30 hours each untreated fruit was 50% rotted, and treatment at this time resulted in 90.2% rot 13 hours later (Fig. 2D) whereas untreated fruits (Fig. 2A) were 100% rotted. The development of aerial mycelium in treated fruits was very sparse.

Effect of Treatment on Nesting: Spread of the fungus by aerial mycelium from diseased to uninjured treated fruits was prevented. A layer of treated fruits was placed in a Petri plate, and fruit from the edge was replaced with inoculated untreated fruit. U-2069 concentrations of 500, 1000, and 2000 ppm were tested, with four replications of each treatment. At 6 days the mycelium extended 44 mm on the untreated fruits (Fig. 3A) but did not extend beyond them onto the fruits treated with 2000 ppm (Fig. 3C). The fungus extended 18 mm onto the fruits dipped in 1000 ppm (Fig. 3B), and 29 mm onto those dipped in 500 ppm.

The above experiment suggested a possible vapor activity of U-2069 against the production of aerial mycelium. Therefore, U-2069 wettable powder was uniformly distributed on the floor of a plastic container. Inoculated Bing cherry fruits were placed on a wire-mesh shelf one inch above the floor. Growth of aerial mycelium was only slight (4 mm turf) in the three containers with the chemical, and abundant (20 mm turf) in containers without U-2069. The differences in aerial mycelium development were more striking than between Figures 3A and 3C.

DISCUSSION

The indicated ability of 2,6-dichloro-4-nitroaniline to control the Rhizopus disease effectively on sweet cherries has great significance to the manner of produce disposition. Control of this disease on other stone fruits could radically change California's marketing program. Furthermore, the information that the chemical shows no effect on respiration of the fruit tissue makes it an ideal material for use in postharvest ripening. With adequate protection on injured areas, mechanical harvest of stone fruits in California will be more feasible.

The activity of this material on a number of isolates of R. stolonifer as well as on other fruit-rot pathogens is under investigation. Control of postharvest rots on apricots and peaches will receive extensive attention. Preliminary studies have also shown promising results with U-2069 concentrations of 100 and 2000 ppm against S. fructicola on sweet cherry fruit, though not as striking.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA

INFECTION OF ARTIFICIALLY INOCULATED SHORTLEAF PINE HYBRIDS WITH FUSIFORM RUST

F. F. Jewell[1]

This paper reports infection of shortleaf pine hybrids following artificial inoculation with southern fusiform rust, Cronartium fusiforme (A. & K.) Hedgc. & Hunt. As shortleaf pine (Pinus echinata) is not considered a natural host (1), it is often crossed with the susceptible slash and loblolly pines (P. elliottii var. elliottii and P. taeda) in breeding for rust resistance. Some success has been reported. Shortleaf x loblolly hybrids have remained rust-free for 5 to 7 years under field exposure (2,4,5). In earlier studies of artificial inoculation no galls developed on shortleaf x slash progenies although foliar symptoms were observed (4,5).

In the present research shortleaf x slash, shortleaf x loblolly, and slash x shortleaf progenies were artificially inoculated on April 28 to May 1 by techniques similar to those reported previously (6). The seedlings were inoculated at 24 days of age, and cotyledons were well developed.

Characteristic symptoms of infection (6) appeared on cotyledons in 2 1/2 weeks, and by 6 weeks after inoculation the affected cotyledons had died on most plants. Symptom expression was uniformly high for all crosses but one (Table 1).

Table 1. Fusiform rust development on inoculated shortleaf x slash and shortleaf x loblolly hybrids.

Parentage ♀ + ♂	Number inoculated	June 13, 1960: number living	June 13, 1960: % with foliar symptoms	November 30, 1960: number living	November 30, 1960: % with galls
Slash-a x shortleaf-a	22	22	95	19	89
Slash-b x shortleaf-b	11	11	91	11	18
Slash-c x shortleaf-b	22	22	41	22	5
Slash-b x shortleaf-a	43	42	93	40	92
Shortleaf-a x slash-a	155	139	100	127	87
Shortleaf-c x loblolly-a	10	9	89	9	22

In November, 7 months after inoculation, typical fusiform galls were present on individuals from each cross (Table 1). Microscopic examination of the gall tissue revealed hyphae and haustoria characteristic of C. fusiforme (3), and pycnia occurred on the hybrids simultaneously with sporulation on slash seedlings that were inoculated at the same time. Apparently this is the first report of the development of fusiform rust galls on hybrids of this type (Fig. 1).

FIGURE 1. Rust-free (A) and rust-infected (B) shortleaf x slash hybrids 9 months after inoculation. Note similarity of galls on B and on slash seedling (C).

[1]Plant Pathologist, Southern Institute of Forest Genetics, Southern Forest Experiment Station, Forest Service, United States Department of Agriculture, Gulfport, Mississippi.

There may be some variation in the reaction of shortleaf to C. fusiforme. Shortleaf parents b and c gave progeny that were considerably less susceptible than those of shortleaf a (Table 1).

Obviously, the mere crossing of any slash or loblolly with the rust-resistant shortleaf will not insure progenies with a high degree of resistance. It appears that inheritance of resistance in interspecies hybrids is more complex than simple dominance, as had previously been assumed (4).

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SOUTHERN FOREST EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, GULFPORT, MISSISSIPPI

RESISTANCE TO POWDERY MILDEW IN LEAFROLL-AFFECTED GRAPEVINES

A. C. Goheen and W. C. Schnathorst[1]

Summary

Symptoms in leafroll-affected grapevines suggested that carbohydrates accumulate in leaves as a result of the virus infection; this was verified by starch and sugar analyses. From this it was predicted that leafroll-diseased vines would be resistant to powdery mildew (Uncinula necator). Counts of mildew colonies on comparable canes of leafroll-affected and leafroll-free vines of the Carignane variety support the prediction that leafroll-diseased vines are resistant to powdery mildew.

Leafroll, a serious virus disease of grapevines (Vitis vinifera) lowers the normal sugar content of the fruit (5). Harmon and Weinberger (6) suggested that this might be due to interrupted translocation of carbohydrates from leaves to fruit in infected vines as a result of phloem necrosis. Leaf symptoms of the disease are rolling and extreme brittleness and in some varieties premature red leaf coloration (5), which suggest carbohydrate accumulation in leaf tissue. Starch and sugars accumulate in the leaves of potato plants infected with potato leafroll virus (1). Potato leafroll is characterized by phloem necrosis, which is partly responsible for the accumulation of carbohydrate.

Accumulation of carbohydrates with accompanying increase in osmotic value of the cell sap is a probable mechanism for resistance to powdery mildews (8, 9). Schnathorst (8) demonstrated that resistance in lettuce (Lactuca sativa) to Erysiphe cichoracearum DC. was correlated with high osmotic value. Weinhold (10) demonstrated a similar correlation in peach (Prunus persica) leaves resistant to Sphaerotheca pannosa Wallr. ex Lév. var. persicae Woron. Delp (3) found that individual grape berries were resistant to Uncinula necator (Schw.) Burr. when the sugar content reached about 8%.

If the leaves of grapevines affected with leafroll are high in carbohydrates, as symptoms suggest, the affected vines may be resistant to powdery mildew. This report describes preliminary observations on carbohydrate concentration and powdery mildew infection of leafroll-affected and leafroll-free grapevines.

STARCH AND SUGAR ANALYSES

Leafroll-affected and healthy Zinfandel vines were established in nutrient culture in the greenhouse. Graft transmission tests to indicator hosts substantiated that leafroll virus was present or absent in the respective vines. Nutrients adequate in potassium (2) were supplied in solution. When symptoms of leafroll were pronounced, comparable leaves from diseased and healthy vines were analyzed for starch and sugar.

For starch analysis several leaves were boiled first 10 minutes in water and then in 95% ethanol until all chlorophyll had been extracted. The leaves were flooded with an IKI solution, and the relative amount of starch was determined by color reaction. Leafroll-affected leaves turned black throughout, whereas healthy leaves were characterized by scattered gray patches. There was no doubt that far more starch was present in leafroll-affected leaves than in healthy leaves.

Sugars were extracted from 3-gram samples of healthy and leafroll-affected leaves. Alcohol extracts of each sample were prepared by blending the tissue in 70% ethanol, boiling 4 minutes in the alcohol, and repeating the extractions. The extracts were concentrated in vacuo at 22° C to final volume of 6 cc, and spotted on Whatman No. 1 filter paper for 1-dimensional descending chromatographic analysis. Chromatograms were developed with 1-butanol-ethanol-water and spots developed with $AgNO_3$. Relative concentration was determined by measuring percentage transmission of the spots with a Photovolt densitometer.

Glucose, fructose, and sucrose were present in extracts of healthy and leafroll-affected leaves. All three sugars were considerably more concentrated in leafroll-affected tissue than in healthy tissue.

Thus, carbohydrate accumulation in leaves appears associated with the leafroll disease of Zinfandel vines. The similarity of disease symptoms indicates that carbohydrate accumulation occurs with leafroll infection in all grape varieties.

[1]Senior Pathologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

RESISTANCE OF LEAFROLL-AFFECTED VINES TO POWDERY MILDEW

The grape variety Carignane is very susceptible to powdery mildew, much more so than Zinfandel, and it shows characteristic symptoms of leafroll in the field. In the Napa Valley of California, incidence of leafroll infection in many Carignane vineyards is 50% or more. In October the contrast between healthy and leafroll vines is marked, and diagnosis of leafroll is as simple as differentiating green from red.

In a Carignane vineyard near Oakville, California, where the vines are planted close together, growth of the canes prevents the grower from practicing powdery mildew control after July. There, powdery mildew annually develops epiphytotically. This vineyard proved an excellent location for comparing the powdery mildew susceptibility of leafroll and healthy grapevines. Eleven stations scattered through the vineyard were selected where leafroll-diseased and leafroll-free vines of the same age stood side by side. The susceptibility of leaves to powdery mildew was compared. In general, leaves of leafroll-free vines appeared more susceptible to powdery mildew than leafroll-affected leaves. However, there was considerable difference in susceptibility to mildew of the leaves on the same vine. The difference apparently depended on the age of the leaves. Tip leaves of leafroll-free vines were highly susceptible, whereas leaves 18 or more nodes back from the tip appeared completely resistant.

Powdery mildew infection of grape canes is much more uniform than infection of the leaves. In vineyards where it is not effectively controlled, infection may be so great on canes of the current season's growth that the entire surface is covered with the powdery mildew fungus. Mildew colonies were counted between the third and tenth nodes on comparable canes of leafroll-affected and leafroll-free vines at the 11 stations in the vineyard. Infection was abundant on the canes of leafroll-free vines, but absent or nearly so on the virus-diseased ones (Table 1, Fig. 1).

Table 1. Mildew colonies between the third and tenth nodes on comparable canes from leafroll-affected and leafroll-free Carignane vines in the same vineyard.

Comparison	Leafroll-affected vines	Leafroll-free vines
1	2	11
2	1	9
3	0	3
4	0	16
5	4	16
6	0	14
7	0	10
8	0	1
9	0	8
10	2	29
11	1	34
Total	10	151

FIGURE 1. Comparable cane sections from leafroll-affected Carignane vine (left) with no powdery mildew infection, and leafroll-free vine (right) with numerous powdery mildew colonies scattered over the bark surface. Internodes are generally somewhat shorter on leafroll canes than on leafroll-free canes.

DISCUSSION AND CONCLUSIONS

This study demonstrates that carbohydrates accumulate in leaves of leafroll-affected grapevines. Carbohydrates probably also accumulate in those areas of the cane that contain chlorophyll, but canes were not analyzed for carbohydrates in this study. Accumulation of carbohydrate in leaves and canes would tend to increase osmotic value, and it is possible that osmotic concentration may be a resistance mechanism, as indicated in studies of Schnathorst (8) on lettuce powdery mildew. In line with this hypothesis, Ravaz (7) observed that red leaves on vines girdled or tightly bound with a ligature had a higher sap concentration than normal leaves and were resistant to U. necator. Although leaves and canes of leafroll vines may be resistant to powdery mildew, the lower sugar content of the berries on these vines may actually make the fruit more susceptible than fruit on a healthy vine.

Gärtel (4) reported that decreasing the potassium supply to the vine increases susceptibility to powdery mildew. Cook and Goheen (2), however, showed that the leaf blades from vines affected with leafroll are deficient in potassium. This contradiction indicates that potassium level per se does not have a direct bearing on powdery mildew resistance. The susceptibility that Gärtel found in potassium-deficient vines is probably an effect of potassium deficiency on chlorophyll content, photosynthesis, and sugar content.

The incidence of leafroll could, and probably does, have an effect on the distribution and severity of powdery mildew in certain grape varieties in some California vineyards.

The reported observations are preliminary. The physico- and biochemical nature of powdery mildew resistance is being investigated further.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA,
DAVIS, CALIFORNIA

THE PATHOGENICITY OF CERTAIN SPECIES OF HELMINTHOSPORIUM TO SPECIES OF THE GRAMINEAE[1]

R. R. Nelson and D. M. Kline[2]

Abstract

The pathogenicity of 12 known and 9 unidentified species of Helminthosporium to 28 species in 22 genera of the Gramineae was determined. In contrast to the concept that most species of Helminthosporium are restricted in pathogenicity in one or a few species, most of the species attacked a wide variety of gramineous hosts. H. sorghicola, H. carbonum, H. maydis, and H. leersiae, each previously reported as pathogenic to one or more species of one genus, were pathogenic to species in 7, 8, 14, and 17 genera, respectively. These studies indicate that the description of new species of Helminthosporium solely on the basis of pathogenicity to a given host is not justified and that certain species described previously on that basis probably should be considered synonymous.

Many species of Helminthosporium attack a wide variety of cultivated and wild species in many genera of the Gramineae. These important pathogens have been associated primarily with leafspot and/or root rot diseases, although some species have been reported as causal agents of seedling blights and diseases of seed and other plant parts. It is commonly believed that many species of Helminthosporium causing leafspot diseases of gramineous hosts are pathogenic only to species in one genus and that most of the remaining species have a limited host range. Many species described on the basis of pathogenicity to specific hosts have never been reported to occur on any other gramineous species (3). This concept of host specificity is widely accepted despite the fact that many species are similar morphologically and difficult, if not impossible, to distinguish from one another solely on the basis of conidial morphology and cultural behavior.

Recent studies have demonstrated cross-fertility between species of Cochliobolus having Helminthosporium conidial stages and between Cochliobolus spp. and species of Helminthosporium with no known sexual stage (2). Hybridization between pathogenic species provides the vehicle for recombination of genes for pathogenicity. Before the pathogenicity of hybrid ascospore progenies can be evaluated, it is necessary to obtain plant species that may serve as differential hosts and to determine their reaction to the parental species of the progenies. The present studies were initiated to determine the reaction of gramineous species to a number of known and unidentified species of Helminthosporium, to select appropriate differential hosts, and to determine whether gramineous species of Helminthosporium are restricted in pathogenicity to one or a few plant species.

MATERIALS AND METHODS

Conidial isolates of 11 species of Helminthosporium obtained from gramineous hosts were used in the present studies (Table 1). One isolate each of 10 species and three isolates of H. turcicum, one each from corn, sorghum, and Sudan grass, were used. One isolate of H. homomorphus, a saprophyte isolated from the air (1), was included as a nonpathogenic control to characterize possible host reactions due solely to the presence of fungus inoculum. In addition, nine isolates of unidentified species of Helminthosporium, two obtained from barley, three from Bermuda grass, and four from oats, were included in the study. The unidentified isolates obtained from one host differed morphologically from one another and, with the exception of H. sp. 1, which is somewhat similar morphologically to H. sativum, also differed from species previously described on that host.

Twenty-eight species in 22 genera of the Gramineae were evaluated for their pathogenic reactions to each of the 23 isolates of Helminthosporium (Table 2). The plants were grown in a 2:1 loam-sand mixture in 4-inch pots in a greenhouse in which the temperature was maintained at

[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the North Carolina Agricultural Experiment Station.

[2]Plant Pathologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Raleigh, North Carolina.

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Table 1. Species of Helminthosporium used in pathogenicity studies.

Species	Source	Number of genera of the Gramineae: reported as hosts[a]	reported hosts used in this study
H. homomorphus Luttrell & Rogerson	Air	-	-
H. maydis Nisik. & Miyake	Corn	1	1
H. carbonum Ullstrup[b]	Corn	1	1
H. sorghicola Lefebvre & Sherwin	Johnson grass	1	1
H. leersiae Atk.	Cut grass	1	-
H. victoriae Meehan & Murphy	Oats	2	1
H. oryzae B. de Haan	Rice	2	1
H. cynodontis Marig.	Bermuda grass	3	2
H. sacchari (B. de Haan)Butl.	Sugarcane	3	1
H. rostratum Drechs.	Corn	5	5
H. turcicum Pass. 1	Corn	5	2
H. turcicum 2	Sorghum	5	2
H. turcicum 3	Sudan grass	5	2
H. sorokinianum Sacc. ex Sorokin	Barley	8	5
H. sp. 1	Barley	-	-
H. sp. 2	Barley	-	-
H. sp. 3	Bermuda grass	-	-
H. sp. 4	Bermuda grass	-	-
H. sp. 5	Bermuda grass	-	-
H. sp. 6	Oats	-	-
H. sp. 7	Oats	-	-
H. sp. 8	Oats	-	-
H. sp. 9	Oats	-	-

[a]Includes only those hosts on which species of Helminthosporium have been reported to cause a leafspot.
[b]Race 1 isolate.

approximately 85° F.

There were considerable differences among species with respect to numbers of plants per pot and height and age of the plants when inoculated. However, the numbers per pot, height, and age of the plants of any given species were comparable in each infection trial. The plants ranged in height from 6 to 18 inches, in age from 10 to 21 days, and in numbers per pot from 1 to 50, depending upon the species used.

The inoculum consisted of a water suspension of mycelium and spores prepared by scraping the surfaces of cultures 7 to 14 days old, grown on potato-dextrose agar in Petri dishes at 75° F. The inoculum was sprayed on the leaves of the plants with a small hand sprayer. The inoculated plants were incubated in a moisture chamber at approximately 75° for 48 hours and then were placed in a greenhouse maintained at 75°.

The plants were examined for disease reactions 10 days after inoculation. The presence of defined lesions on the leaves was the criterion for pathogenicity. The reactions of all plant species to all isolates were confirmed at least once.

RESULTS

In these studies, the pathogenicity of the 12 described species and the 9 unidentified isolates of Helminthosporium to 28 species in 22 genera of the Gramineae is presented in Table 2 and is summarized as follows:

H. homomorphus, the saprophytic species included as a control to detect spurious host reactions, did not incite a lesion reaction on any of the 28 species tested.

H. sorghicola, H. carbonum, H. maydis, and H. leersiae, each previously reported as pathogenic to one or more species of a single genus, were pathogenic to species in 7, 8, 14, and 17 genera, respectively.

H. victoriae and H. oryzae, each reported as pathogens to species in two genera, incited leafspot symptoms on species in 6 and 18 genera, respectively.

H. sacchari and H. cynodontis, reported on species in three genera, and H. rostratum,

Table 2. The pathogenicity of 12 known and 9 unidentified species of Helminthosporium to 28 species in 22 genera of Gramineae.

Species of Helminthosporium[c]	Species of Gramineae and host reaction[a]																												Totals	
	Agrostis palustris	Alopecurus arundinacea	Avena fatua	A. sativa	Axonopus affinis	Bromus catharticus	Cynodon dactylon	Dactylis glomerata	Digitaria sanguinalis	Eleusine indica	Eragrostis curvula	Festuca arundinacea	F. elatior	F. rubra	Hordeum vulgare	Lolium multiflorum	L. perenne	Oryza sativa	Panicum virgatum	Paspalum dilatatum	P. notatum	Pennisetum spicatum	Phalaris arundinacea	P. tuberosa	Poa pratensis	Sorghum sudanense	Triticum aestivum	Zea mays[b]	Species	Genera
H. carbonum	-	-	-	-	-	-	-	L	-	-	-	-	-	-	-	L	-	L	-	-	-	L	-	L	L	L	-	L	8	8
H. cynodontis	-	-	-	-	-	-	L	-	-	-	-	-	-	-	L	-	-	-	-	-	-	-	-	-	-	L	L	-	4	4
H. homomorphus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0	0
H. leersiae	L	L	L	L	L	L	-	L	-	L	L	L	L	L	-	L	L	L	L	-	-	L	L	L	L	L	L	-	22	17
H. maydis	-	L	-	-	L	L	L	L	-	L	L	-	L	L	-	-	-	L	-	-	-	L	-	L	L	L	-	L	15	14
H. oryzae	L	L	L	L	L	L	-	L	-	L	L	L	L	L	L	L	L	L	L	-	-	L	L	L	L	L	L	-	23	18
H. rostratum	-	L	-	-	L	-	-	L	-	L	-	L	L	L	-	-	L	L	-	-	-	L	-	-	L	L	-	L	13	11
H. sacchari	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	L	-	-	-	L	-	L	3	3
H. sorokinianum	L	L	-	-	-	L	-	L	-	-	L	-	L	-	L	L	L	L	-	-	-	L	-	L	-	L	-	-	13	12
H. sorghicola	-	-	-	-	L	-	-	-	-	-	L	-	-	-	-	-	-	L	-	-	-	L	L	L	L	L	-	-	8	7
H. turcicum 1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	L	-	-	-	L	-	L	3	3
H. turcicum 2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	L	-	-	-	L	-	L	3	3
H. turcicum 3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	L	-	-	1	1
H. victoriae	-	-	-	L	-	L	-	-	-	-	-	-	-	-	L	L	-	-	-	-	-	L	-	-	-	L	-	-	6	6
H. sp. 1	-	L	L	-	-	L	-	L	-	L	-	L	-	-	L	L	-	L	L	-	L	L	L	L	-	L	L	-	16	15
H. sp. 2	-	-	L	-	L	L	L	L	-	L	-	L	-	-	L	L	-	L	-	-	-	L	-	-	L	L	-	L	14	14
H. sp. 3	-	-	-	-	L	L	L	-	-	-	-	-	-	-	-	L	-	L	-	-	-	L	-	-	-	L	-	-	7	7
H. sp. 4	-	-	-	-	-	L	L	-	-	L	-	-	-	-	-	L	-	L	L	-	-	L	-	-	-	L	-	L	9	9
H. sp. 5	-	L	-	-	L	L	L	L	-	-	-	-	-	-	L	-	-	L	-	-	-	L	-	-	-	L	L	L	11	11
H. sp. 6	L	L	L	L	-	L	L	L	-	-	L	L	L	-	L	L	L	L	L	-	-	L	L	L	L	L	-	-	20	16
H. sp. 7	L	-	L	L	L	L	-	L	-	L	-	-	-	-	L	-	-	-	-	-	-	-	-	-	-	L	-	-	9	8
H. sp. 8	L	-	L	L	-	L	-	L	-	-	-	-	-	-	L	L	-	-	L	L	-	L	L	L	L	L	-	-	14	12
H. sp. 9	-	-	-	L	L	-	-	-	-	-	-	-	L	-	-	-	-	-	-	-	-	-	-	-	-	L	-	-	4	4
Totals	6	8	7	7	10	13	7	12	0	8	6	6	7	4	10	11	5	13	6	1	1	18	6	9	9	22	5	9		

a-Denotes leaf lesions not produced; L denotes distinct leaf lesion produced.

[b]Inbred line Pr, susceptible to race 1 isolate of H. carbonum, was used in these studies.

[c]Strains of H. turcicum and unidentified species are the same as listed in Table 1.

reported on species in five genera, incited leaf lesions on species in 3, 4, and 11 genera, respectively.

Although H. sativum has been reported on species in 30 genera, it has been reported as a foliage pathogen on species in only 8 genera. In the present studies, H. sorokinianum incited leaf lesions on species in 12 genera.

Isolates of H. turcicum obtained from corn and sorghum caused a leafspot of pearl millet, Sudan grass, and corn. A single isolate of H. turcicum obtained from Sudan grass was pathogenic only to Sudan grass.

The two unidentified isolates of Helminthosporium obtained from barley were pathogenic to species in 14 and 15 genera.

The three unidentified isolates from Bermuda grass incited lesions on species in 7, 9, and 11 genera.

The four unidentified isolates obtained from oats were pathogenic to species in 4, 8, 12, and 16 genera.

In addition to the marked variation in host specificity detected among the various species of Helminthosporium tested, the gramineous species used differed markedly in the numbers of isolates with which a susceptible reaction was obtained. Plants of Sorghum sudanense were susceptible to each of the 22 pathogenic isolates tested, plants of Pennisetum spicatum, Oryza sativa, and Bromus catharticus were susceptible to 18, 13, and 13 isolates, respectively. On the contrary, plants of Digitaria sanguinalis were resistant to each of the 22 pathogenic isolates used, whereas Paspalum dilatatum, P. notatum, Festuca rubra, and Lolium perenne were susceptible to only 1, 1, 4, and 5 isolates, respectively. Species of the remaining genera used fell in between these two extremes in the number of isolates to which they were susceptible.

DISCUSSION

These results demonstrate that species of Helminthosporium are not host specific rather than indicating the potential host range of the species studied. Most fungus pathogens are known to exhibit marked variation in pathogenicity among isolates and, except for H. turcicum, only one isolate of each species was used in these investigations. If additional isolates of the fungus species were used, the numbers of host species exhibiting leafspot reactions to a given species of Helminthosporium probably would have been greater although the reaction of a particular plant species to the various isolates might have been different.

The lack of host specificity among the species of Helminthosporium studied demonstrates that the description of new species solely on the basis of ability to attack a given host is not justified. In these instances, the reaction of a host species to a species of Helminthosporium may represent merely an extension of the host range of a known species. If the practice of describing new species on the basis of host reaction were followed, the two unidentified isolates obtained from barley, which are distinct morphologically and were pathogenic to species in 14 and 15 genera, respectively, could have been described as 29 new species of Helminthosporium.

These results further indicate that some species of Helminthosporium previously described on the basis of pathogenicity to one plant species may not be valid species, but should be considered synonymous. It is not the purpose of this paper to place species into synonymy or to make specific recommendations concerning the revision of the genus Helminthosporium. However, the recognized similarity in morphology of certain species and the fact that these species attack a number of common hosts suggests that a taxonomic study of species in the genus Helminthosporium is needed.

Although the unidentified isolates from one host are different morphologically from one another and from species previously reported as pathogenic to that host, eight of the isolates are similar to other described species. For example, H. sp. 4, isolated from Bermuda grass, is similar morphologically to H. maydis and causes a leafspot of corn indistinguishable from the symptoms caused by H. maydis. In addition, the isolate of H. maydis used caused a leafspot of Bermuda grass similar to that incited by H. sp. 4. It may be concluded that isolate H. sp. 4 is merely a strain of H. maydis which can be differentiated from the isolate of H. maydis used only on the basis of the reaction of other plant species.

Species of 24 of the 42 genera reported as hosts to the 11 pathogenic species of Helminthosporium were used in these investigations (3). In some instances, the host species used was different from the species previously reported as a host to a particular species of Helminthosporium. For example, H. rostratum has been reported to cause a leafspot of Eragrostis cilianensis; E. curvula, however, was used in these tests.

With two exceptions, the isolates of Helminthosporium caused a leafspot on the previously

reported hosts. The isolate of H. cynodontis used was non-pathogenic to Eleusine indica and the isolate of H. sorokinianum studied was non-pathogenic to Triticum aestivum (variety Atlas). Eragrostis curvula, Paspalum dilatatum, and P. notatum were not attacked by H. rostratum, whereas Eragrostis cilianensis and Paspalum boscianum previously had been reported as hosts to that species. These exceptions may be due to variation among strains of H. cynodontis and H. sorokinianum or may be explained by the fact that these two pathogenic species may have been associated with but were not the causal agents of the leafspot diseases.

In addition to single species of 17 genera, two species each of Avena, Lolium, Paspalum, and Phalaris and three species of Festuca were used in these studies. Frequently, isolates of Helminthosporium were pathogenic to one but not to both or to all three of the species of one genus, indicating possible differences in resistance between species. Whenever one species was susceptible and one or more species of the same genus were resistant to an isolate of Helminthosporium, the resistant reactions were confirmed at least twice, a procedure which minimized the possibility that improper inoculation techniques were responsible for the differences between species of a genus.

Since no evidence of infection was observed on plants of any of the 28 gramineous species inoculated with H. homomorphus, this species is considered, within the limits of this study, to be a saprophyte.

As a result of these studies, a number of plant species have been characterized for their disease reactions to the parental isolates of Helminthosporium involved in interspecific crosses. With these differentials it will be possible to evaluate the pathogenic potential of hybrid ascospore progenies and to study the inheritance of genes conditioning pathogenicity in these cross-fertile species.

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DEPARTMENT OF PLANT PATHOLOGY, NORTH CAROLINA STATE COLLEGE, RALEIGH

YELLOW BUD VIRUS ENDEMIC ALONG CALIFORNIA COAST

N. W. Frazier, C. E. Yarwood, and A. H. Gold[1]

Abstract

Peach yellow bud virus has been found occasionally in Fragaria chiloensis, Dipsacus fullonum, Picris echioides, Plantago sp., Stachys bullata, and abundantly in Erigeron glaucus from several coastal localities of California. Identification was principally by symptoms in cucumber and cowpea after mechanical inoculation from native hosts as the native hosts usually were symptomless. Identification by indicator hosts was confirmed by serology. The virus from E. glaucus was additionally confirmed by mechanical inoculation to peach.

Although peach yellow bud mosaic virus (YBV) has a wide host range in artificial inoculations (3, 11), natural infections have been reported only in peach, almond and apricot (8), plum (9), blackberry (1), raspberry and Malva parviflora (3). All are economic Rosaceous hosts except the weed M. parviflora and all have been reported only from inland areas of California but for one record on blackberry in a coastal locality (1).

Collections and assays of wild and cultivated strawberries for other purposes have revealed a few natural infections in native Fragaria chiloensis along the California coast and assay of miscellaneous hosts adjacent to the infected strawberries and at other random sites of similar coastal habitat have shown that Erigeron glaucus is heavily infected in some localities. In one such locality, near Muir Beach, YBV was also recovered from Dipsacus fullonum, Picris echioides, Stachys bullata, and Plantago sp. Most collections were made during winter when plants were matured and the plants from which YBV was recovered did not clearly show symptoms by which they could be distinguished from those that did not yield virus.

E. glaucus was the only host from which YBV was recovered with some degree of consistency and appeared to be a good plant for indicating the coastal distribution of the virus. The results of all assays of this host are presented in Table 1.

Table 1. Collections of Erigeron glaucus assayed for yellow bud virus by mechanical transmission to cucumber and cowpea.

Date and locality of collections		Number of plants assayed	Number of plants infected
Mar. 15, 1960	Noyo	1	1
Mar. 15, 1960	Russian Gulch	5	4
Feb. 5, 1961	Pt. Reyes Lighthouse	8	0
Feb. 5, 1961	Pt. Reyes Beach	9	0
Nov. 11, 1960	Stinson Beach	4	0
Mar. 24, 1960	Muir Beach	8	7
Oct. 21, 1960	Muir Beach	16	9
Dec. 19, 1960	Muir Beach	20	13
Feb. 24, 1961	Muir Beach	33	18
Oct. 31, 1960	San Francisco Beach	14	1
Oct. 31, 1960	Montara	13	0

VIRUS IDENTIFICATION

YBV has been identified principally by transmission to cucumber and cowpea and by serology. While mechanical transmission to peach has been successful (3, 11) it is not considered necessary for routine diagnosis. Cucumber has been the host to which the virus has been most readily transmitted from nature but is not considered to be a diagnostic host because several tree viruses give similar symptoms. But, once YBV has been identified by other methods, cucumber has been a good indicator host. Cowpea has appeared to be the best diagnostic host for mechanical transmission of YBV because it shows symptoms clearly distinct from those of

[1]Entomologist and Plant Pathologists respectively, California Agricultural Experiment Station, Berkeley.

any other virus studied. These symptoms include local lesions on the inoculated leaves, systemic necrosis of the new growth, necrosis of stems below primary leaves, and sometimes an irregular recovery with a distorted type of new growth (3, 11).

One strain of YBV from E. glaucus has been transmitted to peach. In this trial only one out of eight peaches inoculated with the Erigeron virus in cucumber became infected. In this same trial, two out of six peaches inoculated with the virus from peach became infected. The symptoms in peach caused by the virus from Erigeron were milder than the symptoms in peach caused by the virus from peach, but this is not considered significant. There are apparently many strains of YBV, and several isolates from E. glaucus seemed distinct from each other on transfer from cucumber to cucumber.

Serological comparison was used to confirm the relationships between YBV isolates from peach, strawberry and Erigeron which had already been indicated by comparative host symptom response and inoculation experiments. For this purpose, antiserum was prepared against the peach isolate. It was tested by agar gel diffusion reaction against crude juices of cucumber plants that had been infected with the peach, strawberry, and Erigeron isolates. Crude juice of uninoculated cucumber seedlings served as control. All three isolates reacted with the antiserum, but it may be worthy of mention that the Erigeron isolate reacted even more strongly with antiserum than did the peach isolate, whereas the reaction of the strawberry isolate was very much weaker.

DISCUSSION

The occurrence of YBV in widespread localities along the California coast in native wild hosts is evidence that the virus is endemic in that habitat. The apparent tolerance of the host plants to the virus might indicate that this is the natural habitat of the virus rather than the inland areas where the virus has appeared to spread in a few cultivated hosts with symptoms from two orchards in one locality in 1939 (7) to a present distribution in peach in nine counties of northern and southern California (5).

With one minor limitation, Cadman and Lister (2) have presented convincing evidence that tomato ringspot and peach yellow bud mosaic are caused by the same or closely related viruses. If these viruses are the same, the name "tomato ringspot virus" has priority. That we have not used this name requires justification. The one limitation in the evidence of Cadman and Lister is that they do not report that peach yellow bud was produced by inoculation with their tomato ringspot virus. Our virus from Erigeron produces symptoms in peach which is clearly yellow bud mosaic. Additionally, they obtained necrotic lesions and systemic symptoms in tobacco and tomato with both tomato ringspot and peach yellow bud viruses. Our isolates from peach, Erigeron and strawberry have produced only local lesions in both hosts. Inconsistencies in symptom expression of tomato ringspot virus have been noted by Tall, et al. (6) and Price (4) and of peach yellow bud virus by Karle (3). Another disturbing fact is that Cadman and Lister (2) specify 25 mμ as the diameter of their virus particles, whereas Weintraub (10), who secured his virus from the same source, gives 45-50 mμ as the diameter of YBV. Whether the apparent discrepancies noted above justify the retention of the name peach yellow bud mosaic virus remains to be determined.

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DEPARTMENTS OF ENTOMOLOGY AND PARASITOLOGY AND PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

INFLUENCES OF SURFACTANTS AND THE MITICIDE ARAMITE ON CONTROL OF ROSE BLACKSPOT BY PHALTAN

John G. Palmer[1] and T. J. Henneberry[2]

Abstract

At 0.1 percent in a weight to volume ratio six commercial surfactants (two spreaders, one sticker, and three spreader-stickers) were added to multipurpose-spray formulations in all possible combinations with and without the relatively insoluble fungicide N-trichloromethyl thiophthalimide (Phaltan[3]) for control of blackspot and with and without the acaricide 2-(p-tert-butylphenoxy)-1-methylethyl2-chloroethyl sulfite (Aramite[3]) for control of two-spotted spider mites. The experiment was conducted outdoors with two hybrid tea varieties in four blocks of 30 plants with treatments in each block randomized. All plots were sprayed with 1,1,1-trichloro-2,2 bis(p-chlorophenyl) ethane (DDT) and lindane to control chewing and sucking insects. Blackspot and injury ratings were recorded only once. Five conclusions could be drawn: 1) absence of the fungicide resulted in severe blackspot infection followed by defoliation; 2) in two formulations lacking a surfactant, Phaltan with or without Aramite resulted in development of blackspot on only 30% of the rose leaflets; 3) some surfactants significantly decreased control when Aramite was not included; 4) when Aramite was included in a multipurpose formulation containing Phaltan and a surfactant, control of blackspot was significantly increased in most cases; and 5) regardless of the chemicals included in the formulation, the type of foliar injury was the same if any developed.

INTRODUCTION

After experiments at Beltsville indicated that N-trichloromethyl thiophthalimide (Phaltan) controlled blackspot satisfactorily and that no injury resulted from certain multipurpose-spray formulations (1,5), a spray formulation for control of chewing and sucking insects and two-spotted spider mites included Phaltan as one of two fungicides recommended for control of blackspot (4). After distribution of this suggested formulation to a few rose growers during 1959, occasional letters suggested that Phaltan in this formulation or in commercial formulations had injured the leaves of hybrid tea varieties. A few letters mentioned that a surfactant had been included for better control.

Inquiries to commercial formulators revealed that most added a spreader, a sticker, or a spreader-sticker to multipurpose-spray formulations for control of the pests and diseases of roses. In Texas, Lyle (2) reported better control of blackspot by Phaltan when Ortho Spray Sticker[3] (0.5 pint/100 gallons) was included in the formulation. No injury to foliage was reported. When Phaltan at the standard rate of 2 pounds/100 gallons was added to a formulation containing the fungicide maneb and the spreader-sticker Triton B-1956[3], control of blackspot was excellent. Yellowing and burning of foliage were reported, but when the formulation lacked Phaltan injury occurred only if the concentration of other chemicals was increased. At Beltsville, the spreader Santomerse[3] at 0.4 pint/100 gallons (a 1:2000 dilution) had frequently been added to multipurpose formulations including Phaltan with no apparent injury, and this surfactant had been recommended upon request. At Ithaca, New York during 1958, Phaltan at 1 pound/100 gallons combined with a spreader-sticker (probably Ortho Spreader Sticker[3]) at 0.5 pint/100 gallons controlled blackspot and powdery mildew with no chemical injury to foliage as indicated by distortion, discoloration, or burning (3).

Surfactants are considered to be chemically inert and, therefore, little attention is paid to the amount added to a spray formulation in many cases. An "inert" diluent completely suppressed the action of a miticide in a multipurpose-dust formulation for control of rose pests

[1]Formerly Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland. Present address: Forest Disease Laboratory, Forest Service, Beltsville, Maryland.

[2]Entomologist, Entomology Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

[3]Names of chemicals beginning with a capital letter are proprietary. Trade and company names used for identification do not constitute recommendation by the United States Department of Agriculture over others for the same chemical not used.

and diseases (7). In a subsequent study seven such diluents in various commercial preparations variously affected the acaricidal action of malathion and Aramite (6). Since reports of foliar injury attributed to Phaltan often mentioned addition of a surfactant, possible effects of spreaders, stickers, and spreader-stickers upon blackspot control by Phaltan were investigated during the summer of 1960.

METHODS

The experiment was conducted in four blocks of 30 plots each arranged in a randomized-block design. Each plot contained three plants each of the hybrid tea roses Helen Traubel[4] and Red Radiance[4] planted 2 1/2 feet apart, mulched with 1 to 2 inches of aged sawdust, and fertilized uniformly at regular intervals.

All plots were sprayed with the insecticides 1,1,1-trichloro-2,2-bis (p-chlorophenyl) ethane (DDT) (2 pounds of 50% wettable powder formulation/100 gallons) and lindane (1pound of 25% WP formulation) once per week beginning May 16, 1960, to control sucking and chewing insects. Ortho Rose Fungicide[3] (hereinafter called Phaltan) at 1.33 pounds of 75% WP formulation/100 gallons of spray was applied to half of the plots. Each of six commercial surfactants (including the spreaders Santomerse and Tween-20[3], the sticker Ortho Spray Sticker, and the spreader-stickers Triton B-1956, Ortho Spreader Sticker, and Du Pont Spreader Sticker[3] at 0.1% in a weight to volume ratio) was used in all possible combinations with and without Phaltan and the acaricide 2-(p-tert-butylphenoxy)-1-methylethyl 2-chloroethyl sulfite (Aramite) at 2 pounds of the 15% wettable powder/100 gallons of spray in the formulations (See Fig. 1).

Blackspot and injury ratings were recorded on September 16, 1960. Percentage of leaflets on the plants with one or more typical, visible zones of blackspot was recorded by four individuals working independently. Type and severity of injury were recorded for the plots that had been sprayed with formulations containing Phaltan.

RESULTS

Data for blackspot control were analyzed statistically and the LSD's at the 5% level were calculated for both varieties. Because of the similarity of results the data for the variety Helen Traubel are presented in tabular form (Table 1) and for the variety Red Radiance in a column graph (Fig. 1). From these, four conclusions involving control of blackspot are obvious.

Table 1. Control of blackspot by spray formulations with and without each of six surfactants, the miticide Aramite, and the fungicide Phaltan in all combinations applied to field-grown bushes of the hybrid tea rose variety Helen Traubel.

Miticide	Surfactant	Fungicide (Phaltan) added	Fungicide (Phaltan) none
None	None	34[a]	84[a]
None	Santomerse	46	81
None	Triton B-1956	43	85
None	Tween-20	38	86
None	Ortho Spray Sticker	56	80
None	Ortho Spreader Sticker	39	81
None	Du Pont Spreader Sticker	61	85
Aramite	None	30	78
Aramite	Santomerse	26	84
Aramite	Triton B-1956	38	88
Aramite	Tween-20	26	86
Aramite	Ortho Spray Sticker	43	80
Aramite	Ortho Spreader Sticker	36	84
Aramite	Du Pont Spreader Sticker	45	85

LSD at 5% = 11

[a]Percentage foliar blackspot - average of four readings.

[4]Rose plants supplied by courtesy of Armstrong Nurseries, Inc., Ontario, California, and Lone Star Nursery, Tyler, Texas.

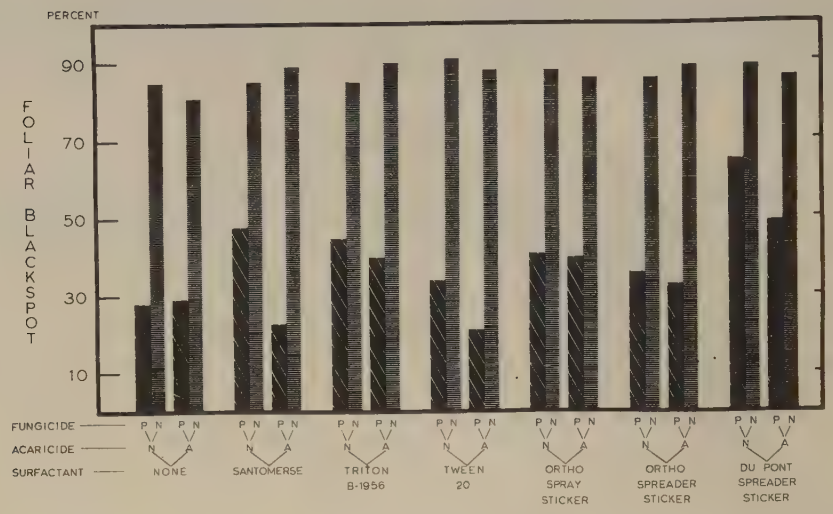

FIGURE 1. Control of blackspot by spray formulations with and without a surfactant, the miticide Aramite, and the fungicide Phaltan in all combinations applied to field-grown bushes of the hybrid tea rose variety Red Radiance. Read from the bottom (surfactant line) upwards. Symbols: P for Phaltan (fungicide), N for none, and A for Aramite (miticide).

(a) Absence of Phaltan resulted in severe blackspot infection. Defoliation was severe all summer, and consequently the bushes were much smaller than those that received multipurpose sprays containing Phaltan. Eighty to 90% of the foliage remaining on the plants had been infected by the blackspot fungus. The acaricide Aramite and each of the surfactants had no significant effect upon control of blackspot in the absence of the fungicide.

(b) In the presence of Phaltan blackspot developed in only 30% of the leaflets of either rose variety if no surfactant was included. Control of blackspot was similar whether Aramite was in the formulation or not.

(c) Addition of a surfactant to a formulation containing Phaltan but not Aramite significantly decreased control of blackspot for all surfactants but Triton B-1956 on the variety Helen Traubel (Table 1) and Tween-20 and Ortho Spreader Sticker on both varieties (Table 1 and Fig. 1).

(d) When the miticide Aramite was included in multipurpose formulations containing Phaltan plus a surfactant, control of blackspot was increased. For the variety Helen Traubel (Table 1) the addition of Aramite to Phaltan plus Santomerse, Tween-20, Ortho Spray Sticker, or Du Pont Spreader Sticker significantly increased control. The increase was not significant for Triton B-1956 and Ortho Spreader Sticker. For the variety Red Radiance (Fig. 1) results were similar except that the addition of Aramite to a formulation containing Phaltan plus Ortho Spray Sticker did not increase control of blackspot.

Lack of Phaltan resulted in severe defoliation, and observations of injury could be made only in plots that had retained foliage through the action of the fungicide. A spotty, water-soaked blackening characterized the injury. Injury ranged from severe (Fig. 2) through medium (for a formulation containing Ortho Spreader Sticker and Aramite) to light (for formulations containing Santomerse). The type of injury was similar regardless of the chemicals included in the formulations. Thus the injury produced by the formulation containing Aramite but lacking a surfactant was similar to that associated with formulations containing surfactants (Fig. 3).

No injury developed when formulations containing Phaltan possessed (a) neither an acaricide nor a surfactant or (b) Ortho Spreader Sticker in the absence of Aramite. However when Aramite was added to the latter formulation, injury resulted that was more severe (rating 1.5) than that induced by Aramite alone (rating 0.5). Very severe injury resulted from use of the spreader-sticker Triton B-1956 and the spreader Tween-20. In these cases the severity was little affected by the presence or absence of Aramite in the formulation. In one case the increase in injury associated with the formulation including Aramite (Tween-20) over that one

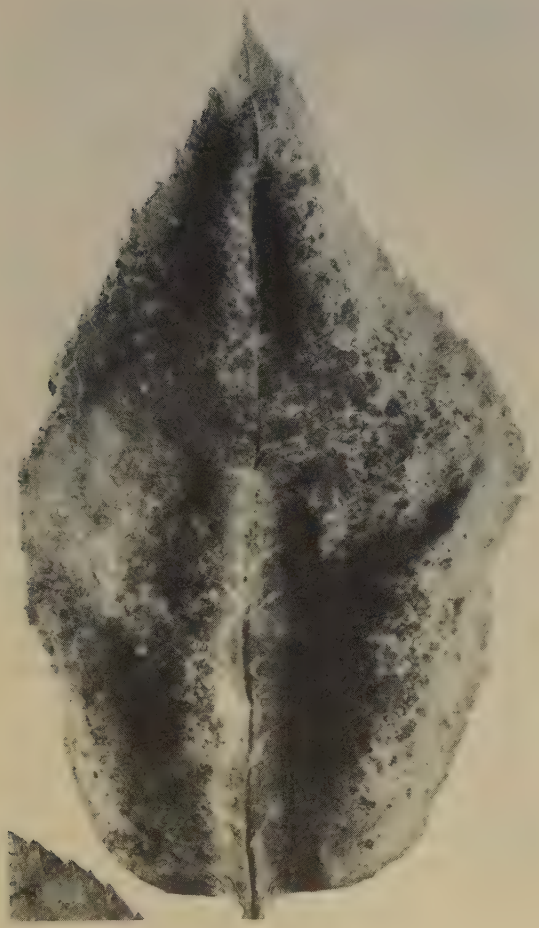

FIGURE 2. Water-soaking type of injury induced by Aramite and several surfactants on leaves of field-grown Red Radiance rose bushes in 1960. Plant was sprayed with a mixture containing the miticide Aramite, the fungicide Phaltan, and the surfactant Du Pont Spreader Sticker.

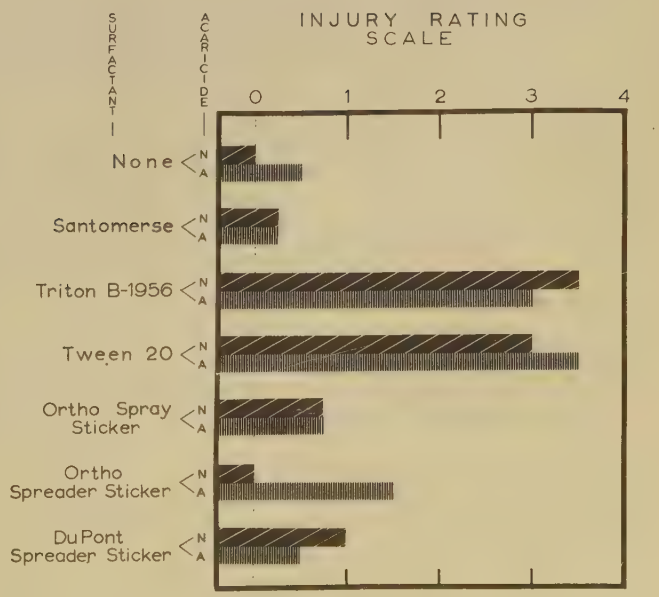

FIGURE 3. Foliar responses in increasing severity from no injury (rating of 0) through injury to 100% of leaflets (rating of 4) on the rose varieties Red Radiance and Helen Traubel in sprays containing six surfactants and Phaltan for control of blackspot plus no acaricide (N) or Aramite (A) for control of mites.

lacking the miticide was similar to that with similar formulations lacking a surfactant. Some injury developed in those formulations in which Aramite was included but no surfactant. No injury occurred when Ortho Spreader Sticker was included in a formulation containing Phaltan but lacking Aramite. The injury that developed from a similar formulation that contained Aramite was greater than the injury attributable to Aramite alone. At least some injury developed from all other formulations. In two instances (Santomerse and Ortho Spray Sticker) Aramite did not influence injury. For Triton B-1956 and Du Pont Spreader Sticker the addition of Aramite reduced foliar injury.

DISCUSSION

Spreaders, stickers, and spreader-stickers influence development of foliar injury on roses and control of blackspot by the water-insoluble fungicide Phaltan when such surfactants are included in a multipurpose-spray formulation for control of sucking and chewing insects, mites, and blackspot. In general injury was more pronounced on foliage of Helen Traubel than on foliage of Red Radiance. Injury developed because the dosages of surfactants were heavy, but the association of Aramite with development of injury on the foliage was unexpected.

The general reduction in control of blackspot by formulations containing a surfactant as compared with the spray lacking them was not unexpected. No influence on control of blackspot by Ortho Spreader Sticker was expected since this surfactant is manufactured by the firm that produces Phaltan.

The role of Aramite, its diluent, or the combination in improving control of blackspot by formulations containing a surfactant and the fungicide compared with similar formulations lacking Aramite is baffling. The most significant increases in blackspot control on both rose varieties were recorded when Aramite was added to Phaltan plus Santomerse, Tween-20, or Du Pont Spreader Sticker, but a significant increase in control developed with Ortho Spray Sticker (a product of the manufacturer of Phaltan) on the variety Helen Traubel only. A greater reduction in control of blackspot through more rapid solution of the fungicide and subsequent removal from the rose leaflet or concentration around the periphery and in vein depressions was anticipated with use of spreaders. This failed to materialize and, in fact, incidence of blackspot was less than in the controls upon the addition of Aramite to formulations containing the fungicide plus Santomerse or Tween-20. If Aramite, its diluent, or the combination of these triggered the response, a change in either might eliminate the response. Were a different fungicide or diluent to be used, possibly the response would be altered or would not occur in the presence of Aramite and a surfactant. Indeed a change in the diluent of one of the insecticides or of the fungicide could influence the responses.

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CROPS RESEARCH AND ENTOMOLOGY RESEARCH DIVISIONS, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND

REACTION OF WHEAT SEEDLINGS TO NEW ISOLATES OF WHEAT STEM RUST[1]

James D. Miller and Donald M. Stewart[2]

New and potentially important isolates of stem rust (Puccinia graminis Pers. f. sp. tritici Eriks. & E. Henn.) have been found in the past 2 years in the physiologic-race survey and in rust collected in rust nurseries in the United States and Mexico. The most important of these isolates were identified as races 11, 15, 15B, the 17-29 group, 32, 34, and 48A. Pathogenicity of 37 isolates of these races was tested on seedlings of many lines and varieties of wheat. Thirty of these isolates were tested on the supplemental differentials: 63% of the 30 isolates were virulent on Selkirk, 80% on Golden Ball, 43% on Bowie, 3% on Kenya Farmer, and 23% on Frontana-K58-Newthatch.

A composite of the 37 isolates was used to inoculate seedlings of 286 spring wheats, including advanced selections, potential parental material, and named varieties at greenhouse temperatures of 18° and 29° C. Thirty-eight percent of the 286 wheats were resistant and 16% were susceptible to the composite at 18°, whereas the other 46% were resistant to some components of the inoculum and susceptible to other components. At 29°, on the other hand, 19% of the lines were resistant and 25% were susceptible to the composite, whereas 56% were resistant to some components and susceptible to others. If all wheats with susceptibility to all or a part of the composite are grouped at one or at both temperatures, 85% of the 286 wheats might be inadequately protected if the new rust cultures increased in prevalence. The other 15% consistently were resistant at both temperatures and merit further study.

Ten of the 286 wheats are of immediate concern in the spring wheat area and their reactions to the composite were determined in somewhat greater detail. The ten comprised three common wheat varieties, Selkirk, Pembina, and Canthatch, four durum wheat varieties, Wells, Lakota, Langdon, and Ramsey, and three promising Minnesota common wheat selections, II-53-562 and II-53-567 selected from K58-Newthatch x Lee[3] and II-53-404 selected from Klein Titan-Thatcher[3] x (K58-Newthatch x Thatcher[2]). Three of the ten performed exceedingly well; II-53-404, Wells, and Lakota were resistant to the composite at both temperatures (Table 1). Forty percent or more of the infections that developed on Selkirk, II-53-562, Langdon, and Pembina at both temperatures were in the susceptible class (types 3 and 4). However, on Selkirk and Pembina there were varying amounts of chlorosis and necrosis surrounding the uredia. Ten to

Table 1. Percentage of pustules in the resistant and susceptible classes on ten spring wheat varieties and selections inoculated with a composite of 37 isolates of Puccinia graminis f. sp. tritici at 18° and 29° C.

Variety or Selection	C.I. No.	Temperature, reaction class, and percentage of pustules in each class[a]			
		18° C		29° C	
		resistant class	susceptible class	resistant class	susceptible class
		%	%	%	%
Lakota	13335	100	0	100	0
Wells	13333	100	0	100	0
II-53-404	13415	100	0	100	0
II-53-567	13416	90	10	30	70
Canthatch	13345	80	20	10	90
Ramsey	13246	80	20	10	90
II-53-562	13458	50	50	50	50
Selkirk	13100	30	70	50	50
Langdon	13165	20	80	60	40
Pembina	13332	20	80	10	90

[a]A 50 in the resistance class means that 50% of the lesions were any one or a combination of infection types 0;, 1, and 2. A 50 in the susceptibility class means that 50% of the lesions were any one or a combination of infection types 3 and 4.

[1]Investigations conducted in cooperation with the Minnesota Agricultural Experiment Station. Paper No. 4600, Scientific Journal Series.

[2]Plant Pathologists, Crops Research Division and Plant Pest Control Division, respectively, Agricultural Research Service, United States Department of Agriculture, St. Paul, Minnesota.

Table 2. Seedling reaction of ten spring wheat varieties and selections to two isolates of Puccinia graminis f. sp. tritici race 15B at 18° and 29°C.

Variety or Selection	C.I. No.	Isolate, temperature, and varietal reaction			
		R 15B[a]		R 15B[b]	
		18°C	29°C	18°C	29°C
Lakota	13335	R[c]	R	R	R
Wells	13333	R	R	R	R
II-53-404	13415	R	R	R	R
Ramsey	13246	R	R	R	S
Pembina	13332	R	S	MS	S
Selkirk	13100	R	S	MS	S
Canthatch	13345	S	S	MS	MR
Langdon	13165	S	S	MS	S
II-53-562	13458	S	S	R	S
II-53-567	13416	S	S	R	S

[a]Race 15B obtained from Langdon durum in Minnesota, 1959. Culture no. 59-21-60.

[b]Race 15B obtained from Selkirk in Texas, 1959. Culture no. 59-41-23.

[c]S = susceptible, MS = moderately susceptible, MR = moderately resistant and R = resistant.

20% of the infections on II-53-567, Ramsey, and Canthatch were in the susceptible class at 18°C, whereas 70 to 90% of the infections were in the susceptible class at 29°. Races 15B and 29 were recovered from infection types 3 and 4 on Selkirk, Pembina, and Canthatch and race 15 from Selkirk.

The ten varieties and selections again were tested with two isolates of race 15B isolated from Selkirk and Langdon, the predominant wheats in the spring wheat area. The seedling reactions of the ten wheats to the two isolates of race 15B at 18° and 29°C are given in Table 2. Wells, Lakota, and II-53-404 were resistant to both isolates at both temperatures, and Ramsey was susceptible only to the isolate from Selkirk at 29°. Of the remaining six varieties and selections, Selkirk and Pembina were resistant to the Langdon isolate and moderately susceptible to the Selkirk isolate at 18°, II-53-562 and II-53-567 were resistant to the Selkirk isolate at 18°, Canthatch was moderately resistant to the Selkirk isolate at 29°, and Langdon was moderately susceptible to the Selkirk isolate at 18°. Adult plant reactions of these ten varieties and selections at 24° generally were similar to their seedling reactions at 18°. The exceptions were the moderate susceptibility of Pembina adults to the Langdon isolate and the susceptibility of adults of both Selkirk and Canthatch to the Selkirk isolate.

These results indicate that races of the stem rust fungus occur in the United States that are a potential threat to the commercial spring wheat varieties grown today. However, the two new durum varieties, Lakota and Wells, and Minnesota selection II-53-404 appear to be resistant to these stem rust isolates.

CROPS RESEARCH AND PLANT PEST CONTROL DIVISIONS, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, ST. PAUL, MINNESOTA

SUSCEPTIBILITY OF DETACHED ROYAL APRICOT FRUITS TO VARIOUS PLANT PATHOGENS

Thomas H. Stanton and Joseph M. Ogawa[1]

Information on the susceptibility of apricot (Prunus armeniaca) fruits to infection by microorganisms will aid in identification of various symptoms and signs found associated with stored fruits. In this study plant pathogens commonly occurring in orchards, as well as those not associated with orchard crops, were tested. Comparisons were made with: 1) Known pathogens of the apricot tree. Sclerotinia (Monilinia) fructicola (Wint.) Rehm, S. laxa Ader. & Ruhl., which are responsible for much of the postharvest decay of apricot fruit in California (2). Alternaria citri Ell. & Pierce (A. tenuis sensu Neerg.) causing a blossom end rot of apricot fruits (3). Botrytis cinerea Pers. ex Fr. (green fruit rot), Coryneum beijerinckii Oud. (shot hole on leaves and lesions on fruits), Verticillium albo-atrum Reinke & Berth. (black heart), Pseudomonas syringae Van Hall (bacterial canker), Cytospora sp. (dieback), Armillaria mellea Vahl. ex Fr. (root rot), and Cytosporina sp. (dieback). 2) Pathogens of other stone-fruit species. Sclerotium rolfsii Sacc. (stem rot of peach seedlings), Aspergillus niger van Tiegh. (black mold on peach), Ceratocystis fimbriata Ell. & Halst. (mallet wound canker of almond). 3) Pathogens that have not been reported to produce diseases of stone-fruit trees. Phomopsis cinerascens (Sacc.) Tav. (branch canker of fig), Phytophthora syringae Kleb. (lilac blight), Penicillium digitatum Sacc. (green mold of citrus), Gibberella fujikuroi (Saw.) Wr. (pink rot of corn), Septoria apii (Briosi & Cav.) Chester (late blight of celery), Pythium ultimum Trow (damping-off of sugar beets), and Pellicularia filamentosa (Pat.) Rogers (damping-off of sugar beets).

Harvest-mature Royal apricot fruits were surface sterilized with 1% Clorox (5.25% sodium hypochlorite) solution for 5 minutes and rinsed in distilled water. A small piece of mycelium was taken from cultures grown on 1% potato-dextrose agar and placed in a 5-mm stab made with a pointed dissecting knife to the stylar end of three fruits. Fruits were then incubated at 24° C in nonairtight plastic containers. Data were taken 7 days after inoculation when about 30% decay resulted with S. fructicola inoculation.

The degree of fruit decay was as follows: 1) 25 to 100%; B. cinerea, Phytophthora syringae, S. rolfsii, P. digitatum, A. niger, and A. tenuis. 2) less than 25%; C. beijerinckii, Cytosporina sp., V. albo-atrum, G. fujikuroi, Pseudomonas syringae, P. cinerascens, and C. fimbriata. 3) zero; S. apii, P. ultimum, Cytospora sp., A. mellea, and P. filamentosa.

Another test was made on five harvest-ripe Royal apricot fruits which were inoculated as above and incubated for 6 days. The average percentage decay on a flesh weight basis was: S. rolfsii 61.7, Phytophthora syringae 95.1, G. fujikuroi 13.1, and C. beijerinckii 8.5. The uninoculated fruits showed no decay.

Decay was also produced on Elberta peach (Prunus persica) with S. rolfsii, Phytophthora syringae, and C. beijerinckii. The ability of C. beijerinckii to produce fruit decay has previously been reported for cherry (Prunus avium) (1) and peach (4).

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA

[1]The senior author, a high school student from La Sierra High School, Carmichael, California, was a participant in the National Science Foundation summer school program in Microbiology for Talented Secondary School Students.

FOMES ANNOSUS CAUSES SEVERE DAMAGE TO THINNED SLASH PINE PLANTATION IN SOUTH GEORGIA

Charles H. Driver and T. R. Dell

Recent reports indicate that Fomes annosus (Fr.) Cke. is widespread in the south and at times causes severe loss in thinned slash pine (Pinus elliottii var. elliottii) plantations[1, 2]. However, few accounts give specific volume losses and information on the rapidity with which these losses develop following thinning. An opportunity to obtain such data was afforded by a severely diseased stand on the Southlands Experiment Forest in Decatur County, Georgia.

The affected 1.7-acre stand was planted in 1938 as a 1000 x 75 foot game cover strip between cultivated fields. Spacing was irregular, with rows averaging 11 feet apart and trees 3 feet apart within the row. The cultivated fields were planted in 1948 to slash pine and hence the original 1.7-acre strip, now 23 years of age, is surrounded by 200 acres of 12-year-old plantation. The root rot history of the original strip planting is particularly interesting in view of the 2000 acres of 12-year-old plantations in the Southlands Experiment Forest now being thinned for the first time.

The original planting was thinned during the summer of 1956 when the stand was 17 years old. This thinning removed 12 cords per acre. Five years later three conspicuous openings with many dead dying trees had developed in the stand. Fomes annosus was fruiting in abundance on both dead standing and still living trees.

Data on the stand volume showed that 40 cords per acre were in living trees. However, still living trees to the extent of 3 cords per acre had root rot symptoms. Six cords per acre were in dead standing or fallen stems.

In a reconstruction of the stand, and a disregard for growth losses on diseased and living trees, 5 years after the original thinning the stand should have contained approximately 46 cords per acre. However, owing to 6 cords already dead and 3 more diseased and dying, the 5-year loss following thinning was approximately 20% of the stand. These losses are still continuing and further data will be taken to determine rate of spread as the stand ages.

Data taken over the most abundant fruiting periods within the past year on the presence or absence of fruiting bodies on stumps of different sizes are presented in Table 1.

Table 1. Diameter of slash pine stumps in relation to fruiting of Fomes annosus.

Stump diameter 2-inch diameter classes	Total number stumps	Fruiting bodies present	
		number	%
4	17	0	0
6	162	5	3.1
8	175	8	4.6
10	92	10	10.9
12	37	5	13.5
14	3	1	33.3

The data apparently indicate that the larger the stump the greater the likelihood of infection and fruiting by Fomes annosus.

SOUTHLANDS EXPERIMENT FOREST, INTERNATIONAL PAPER COMPANY, BAINBRIDGE, GEORGIA

[1]Driver, Chas. H. and T. R. Dell. 1961. Fomes annosus root-rot in slash pine plantations of the Eastern Gulf Coast States. Plant Disease Reptr. 45: 38-40.

[2]Powers, H. R. Jr., and John S. Boyce, Jr. 1961. Fomes annosus on slash pine in the South east. Plant Disease Reptr. 45: 306-307.

DISTRIBUTION AND PREVALENCE OF PHYSIOLOGIC RACES OF LEAF RUST OF WHEAT, PUCCINIA RECONDITA ROB. EX DESM., IN THE UNITED STATES IN 1960[1]

C. O. Johnston[2]

Summary

Only 175 field collections (about 30%) of leaf rust of wheat made during 1960 yielded cultures for physiologic race determinations. These, representing collections from 22 States, yielded 522 single-pustule isolates from which 16 races were determined. Physiologic race 15, followed by races 5 and 122, was the most prevalent and widely distributed.

A large number of field collections of leaf rust of wheat, Puccinia recondita Rob. ex Desm., were received in 1960, but cultures were obtained from only 175, which was less than one third. For the first time in many years many of the collections were nonviable, probably because of unfavorable conditions during shipment and storage. No cultures were obtained from collections made in States along the Pacific Coast or in the intermountain area. Furthermore, no cultures were obtained from collections from such important wheat-growing States as Nebraska, Colorado, Indiana, Kentucky, and Tennessee. In many instances where infection was obtained only a few uredia developed.

The 175 collections cultured yielded 522 single-pustule isolates from which 16 races were determined for that part of the United States east of the Rocky Mountains. These came from cultures obtained from collections from the 22 States shown in Table 1.

Physiologic race 15 was the most abundant and widely distributed race in the United States for the fourth consecutive year. It comprised 44.4% of all isolates and appeared in collections from all States except Ohio and Maryland.

Race 5 was second in importance, comprising 15.7% of all isolates but was not so uniformly distributed as race 15. It was especially abundant in Illinois, Florida, Kansas, and Minnesota.

Race 122 was third in prevalence, comprising 11.9% of all isolates. It apparently was more widely distributed than race 5, appearing in isolates obtained from collections made in 12 of the 22 States. It was especially abundant in collections from Michigan, Virginia, and Kansas.

Race 9 was fourth in abundance, comprising 6.7% of all isolates. Unified race 9, made up of the similar races 9, 19, and 20, represented 13.2% of all isolates. Race 9, for many years prior to 1948 the dominant one in central United States, appears to be increasing in prevalence again.

Race 58, long the dominant race in northeastern United States, especially in the Ohio River Valley and around the Great Lakes, was important in Illinois, Ohio, and New York.

No new or unusual races were isolated from 1960 collections. A few races appeared to be rather rare. Race 52 was isolated only once, race 1 twice; and races 2 and 35 were isolated three times each.

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[1]Contribution No. 578, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, in cooperation with the Crops Research Division, Agricultural Research Service, United States Department of Agriculture. Botany serial No. 740.

[2]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

Table 1. Physiologic races of the leaf rust of wheat, Puccinia recondita Rob. ex Desm., isolated from collections made in the United States during the spring, summer and fall of 1960.

Geographic and crop areas and states	Number of isolates of unified races and constituent physiologic races																	Total Number		
	1	2		3	5		6		9			10	13				Isolates	Races	Collections
	1	2	15	58	5	52	6	105	9	19	20	11	35	54	77	122			
Northeastern - Soft Red Winter																			
Arkansas			8		1				3				1			3	16	5	5
Illinois			8	3	10		4	1	2		1				1	6	36	9	12
Michigan			3													9	12	2	1
Missouri			8		3					2	1	1				1	16	6	7
New York			1	7			1							4			13	4	3
Ohio				4				2						6			12	3	3
Pennsylvania			4		2							2		1		4	13	5	4
Southeastern - Soft Red Winter																			
Alabama			3				4									1	8	3	8
Florida			7		11									1		1	20	4	5
Georgia			3													1	4	2	3
Maryland				1			4										5	2	3
North Carolina	1		3														4	2	3
Virginia			12		7										2	11	32	4	7
West Virginia			1														1	1	1
Central - Hard Red Winter																			
Kansas	1	2	94	2	22				20	12	12	2	1	4		21	193	12	57
Oklahoma			3						3								6	2	3
Texas			4		2		2		3	1	2	2	1	1	2		20	10	10
Wyoming			2		2											2	6	3	1
North Central - Hard Red Spring																			
Iowa			24		7	1		1	2	3					1	2	41	8	15
Minnesota		1	37		13		1										52	4	19
North Dakota			5		2				2					1			10	4	3
South Dakota			2														2	1	2
Total	2	3	232	17	82	1	16	4	35	18	16	7	3	18	6	62	522	16	175
Percentage of total isolates	0.4	0.6	44.4	3.3	15.7	0.2	3.1	0.8	6.7	3.4	3.1	1.3	0.6	3.4	1.1	11.9			

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, KANSAS STATE COLLEGE, MANHATTAN IN COOPERATION WITH CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

STUDIES OF THE EFFECT OF STRAW COVER AND METHOD OF INOCULATION ON DWARF BUNT OF WINTER WHEAT IN UTAH

Wade G. Dewey[1]

Summary

The comparative effects of straw cover versus no cover and spray versus soil inoculation on the incidence of dwarf bunt in winter wheat were studied in 1958 and 1960. A highly significant difference in favor of soil inoculation was observed both years. Straw-covered plots showed significantly less dwarf bunt in 1958 but significantly more bunt in 1960 than did non-covered plots.

INTRODUCTION

Possibly the greatest deterrent to progress in breeding wheat varieties resistant to the dwarf bunt organism Tilletia contraversa Kuehn, has been the inability of the breeder to induce sufficiently high levels of infection in test nurseries to permit the reliable separation of resistant and susceptible segregates. Numerous attempts to develop inoculation methods that would produce consistently high infection under field conditions have met with varied success; however, no generally applicable method has as yet been forthcoming.

Notable among recent techniques that have increased smut percentages in certain areas where dwarf bunt is a problem are the use of infested soil followed by a straw covering, reported by Tyler and Jensen (5, 6) in New York, and shallow planting followed by spray inoculation of the soil surface, as reported by several workers from the Pacific Northwest (3, 4).

Inasmuch as dwarf bunt is a potentially serious disease problem in Utah, studies have been undertaken to test the applicability of straw cover and spray inoculation as methods of increasing the incidence of dwarf bunt under Utah conditions. This paper reports the findings of tests conducted in 1958 and 1960.

METHODS

Twenty-five replications of straw cover versus no cover treatments were completely randomized over plots four rows wide and 5 feet long. Spray versus soil inoculation treatments were randomized on halves of each main plot, giving 50 replications of each sub plot treatment. The same experimental design was used in both the 1958 and the 1960 studies.

Wheat was sown by hand in open furrows and either covered with infested soil to a depth of 1/2 to 1 inch or covered to this depth with non-infested soil and the surface sprayed with a heavy spore suspension. The soil used for the soil inoculation treatment was prepared by mixing 100 grams of coarsely ground spore material (entire infected heads were ground in an ordinary meat grinder) with 1 bushel of soil. The infested soil was exposed to weathering in shallow, open bins for periods of 1 week and 3 1/2 weeks prior to application in the 1958 and 1960 tests, respectively. Approximately 1/2 gallon of infested soil was used to cover each 5-foot row.

The spray-inoculated plots were sprayed with a suspension of spores that had been prepared by mixing finely sieved spore material with water and filtering the suspension through several layers of cheesecloth. The spore suspension was applied with a 3-gallon sprayer. Rows were sprayed both before and after planting and an attempt was made to apply approximately the same quantity of spore material by both the spray and soil inoculation methods.

Spores for the 1958 trial were collected at Nephi, Utah in 1954 and presumably represented the D-1 race of dwarf bunt which has been particularly prevalent in that area. Spores used in the 1960 test were collected near Snowville, Utah, in 1958 and have been identified by the Regional Smut Research Laboratory at Pullman, Washington, as the more virulent D-3 race.

The highly susceptible variety, Hybrid 128, was used in 1958. Itana, a variety relatively susceptible to race D-3, was used in 1960.

Plots receiving straw cover were covered to a depth of approximately 3 inches early in December. The straw was removed during the first week in April. Infection percentages were computed from the ratio of infected tillers to total tillers in each plot.

RESULTS

Tables 1 and 2 summarize the results of the 1958 and 1960 tests. In both years differences

[1]Associate Professor, Department of Agronomy, Utah State University, Logan, Utah.

Table 1. Percentage of dwarf bunt infection in winter wheat plots receiving straw cover versus no cover and spray versus soil inoculation. 1958 test.

Straw-covered main plots				Non-covered main plots			
Plot no.	Soil-inoculated sub plot	Spray-inoculated sub plot	Average main plot infection	Plot no.	Soil-inoculated sub plot	Spray-inoculated sub plot	Average main plot infection
1	25.5	29.4	27.5	3	33.1	26.1	29.6
2	25.3	22.8	24.1	4	33.3	21.1	27.2
5	35.1	17.5	26.3	6	44.2	27.5	35.9
8	17.4	23.6	20.5	7	35.5	24.4	30.0
10	15.2	15.7	15.5	9	35.2	45.5	40.4
12	5.8	18.4	12.1	11	39.1	43.6	41.4
13	24.5	10.5	17.5	14	56.6	28.5	42.6
15	4.5	20.2	12.4	18	31.0	37.6	34.3
16	37.0	19.6	28.3	23	41.8	35.2	38.5
17	11.5	21.7	16.6	25	41.0	35.1	38.1
19	18.0	24.0	21.0	27	40.5	38.9	39.7
20	73.9	25.4	49.7	28	49.0	47.3	48.2
21	39.4	5.9	22.7	30	54.7	37.9	46.3
22	30.4	24.2	27.3	31	44.5	54.0	49.3
24	31.7	14.0	22.9	33	47.8	31.9	39.9
26	24.1	14.5	19.3	35	56.3	38.9	47.6
29	32.6	41.1	36.9	38	45.5	22.3	33.9
32	13.1	32.1	22.6	39	72.2	35.8	54.0
34	8.8	6.6	7.7	40	45.2	26.8	36.0
36	15.2	1.6	8.4	41	39.4	37.2	38.3
37	26.7	3.3	15.0	42	45.0	39.9	42.5
43	10.4	7.8	9.1	44	52.3	23.2	37.8
45	12.7	16.1	14.4	46	40.2	12.9	26.6
48	43.5	16.2	29.9	47	30.1	48.3	39.2
49	26.3	23.5	24.9	50	38.6	31.8	35.2

		% infection
Main treatment means:	Straw-covered plots	21.3
	Non-covered plots	38.9
Sub plot means:	Soil-inoculated plots	34.0
	Spray-inoculated plots	26.1
Means for main and sub plot combinations:	Straw cover - soil inoculation	24.3
	No cover - soil inoculation	43.7
	Straw cover - spray inoculation	18.2
	No cover - spray inoculation	34.1

Table 2. Percentage of dwarf bunt infection in winter wheat plots receiving straw cover versus no cover and spray versus soil inoculation. 1960 test.

Straw-covered main plots				Non-covered main plots			
Plot no.	Soil-inoculated sub plot	Spray-inoculated sub plot	Average main plot infection	Plot no.	Soil-inoculated sub plot	Spray-inoculated sub plot	Average main plot infection
1	31.3	5.0	18.2	3	22.7	12.1	17.4
2	20.9	34.7	27.8	4	15.4	4.5	10.0
5	17.7	17.0	17.4	6	25.1	5.0	15.1
8	38.2	17.7	28.0	7	28.4	4.9	16.7
10	23.5	19.1	21.3	9	35.4	5.2	20.3
12	24.9	24.0	24.5	11	31.3	9.3	20.3
13	41.5	21.2	31.4	14	17.0	5.9	11.5
15	30.3	15.9	23.1	18	23.6	10.0	16.8
16	35.8	14.5	25.2	23	20.5	4.7	12.6
17	24.7	10.2	17.5	25	29.8	5.5	17.7
19	36.7	18.3	27.5	27	20.6	2.7	11.7
20	38.8	5.9	22.4	28	24.8	5.8	15.3
21	44.8	8.0	26.4	30	22.9	6.7	14.8
22	32.1	43.5	37.8	31	28.9	4.1	16.5
24	54.6	9.5	32.1	33	22.4	3.8	13.1
26	41.4	3.6	22.5	35	35.4	4.0	19.7
29	15.0	16.7	15.9	38	31.5	6.4	19.0
32	14.8	5.1	10.0	39	22.2	11.9	17.1
34	18.5	16.5	17.5	40	8.8	4.1	6.5
36	31.5	10.6	21.1	41	10.4	4.2	7.3
37	23.8	10.4	17.1	42	23.3	5.7	14.5
43	17.5	12.2	14.9	44	27.3	7.2	17.3
45	11.0	9.3	10.2	46	16.7	6.8	11.8
48	17.0	6.0	11.5	47	18.3	4.0	11.2
49	27.2	6.1	16.7	50	15.6	13.0	14.3

		% infection
Main treatment means:	Straw-covered plots	21.5
	Non-covered plots	14.7
Sub plot means:	Soil-inoculated plots	25.8
	Spray-inoculated plots	10.4
Means for main and sub plot combinations:	Straw cover - soil inoculation	28.5
	No cover - soil inoculation	23.1
	Straw cover - spray inoculation	14.4
	No cover - spray inoculation	6.3

between main plot treatments and between sub plot treatments were highly significant. There were no significant interactions between main and sub plot treatments.

The 1958 results were opposite to those reported from New York with respect to straw cover, and opposite to those from the Pacific Northwest with respect to spray inoculation. Infection percentages in the open plots nearly doubled those recorded in straw-covered plots. A highly significant increase in dwarf bunt infection was also observed in soil-inoculated plots when compared with those receiving spray inoculation. The no cover-soil inoculation combination of main and sub plot treatments proved most effective, resulting in more than twice as much bunt as the straw cover-spray inoculation combination. The no cover-spray inoculation and the straw cover-soil inoculation combinations were intermediate in their effect upon bunt expression.

Results of the 1960 trial were similar to those in 1958 with respect to soil versus spray inoculation. Average infection percentages in soil-inoculated plots were more than double those observed in spray-inoculated plots. The relative effectiveness of straw cover versus no cover, however, was the reverse of that noted in 1958. Infection in the covered plots averaged 50% higher than in the open plots. The most effective combination of main and sub plot treatments in 1960 was straw cover-soil inoculation, which resulted in approximately four times as much bunt as the no cover-spray inoculation combination.

DISCUSSION

Divergent results from area to area and even from year to year within the same area characterize investigations involving the dwarf bunt organism and tend to emphasize the dependency of dwarf bunt expression on environmental factors.

The apparent superiority of soil inoculation over spray inoculation in the studies thus far conducted in Utah is in contrast to findings by workers in the Pacific Northwest (3, 4) and is somewhat disappointing inasmuch as spray inoculation is considerably easier to use. A possible explanation for the realtively low infection resulting from spray inoculation might be associated with poor surface moisture in the fall. This could conceivably put surface spores at a germination disadvantage compared with spores incorporated into the upper inch of soil. Both Baylis (1, 2) and Meiners, et al. (4) have reported good germination and infection when spores were broadcast or sprayed on a moist soil surface, and delayed germination and relatively poor infection when spores were applied to a soil surface that remained dry for an extended period. Since a dry soil surface is generally characteristic of fall conditions in the Intermountain region, soil inoculation may have a certain inherent advantage in this area. Studies using several modifications of the spray inoculation method are being continued in an attempt to develop a spray procedure that will prove equal or superior to soil inoculation in Utah.

The possible value of straw cover as a means of increasing dwarf bunt infection under Utah conditions is as yet undetermined. Conflicting results between the 1958 and 1960 tests indicate, however, that the method is at least less reliable in this area than it appears to be in New York. Since straw cover undoubtedly exerts its influence by modifying the micro-environment to which the plants and the pathogen are exposed, it could be expected that variation in the macro-environment due to major geographical and climatic changes might modify the influence of straw cover from one area to another. Investigations into the use of protective cover are being continued.

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DEPARTMENT OF AGRONOMY, UTAH STATE UNIVERSITY, LOGAN, UTAH

OCCURRENCE OF CERCOSPORA BOUGAINVILLEAE IN EL SALVADOR

Oscar Ancalmo[1]

Cercospora bougainvilleae Muntañola[2] was found for the first time in El Salvador in 1960. Conidia were present on leaves of bougainvillea (Bougainvillea glabra Choisy) collected in the San Salvador area in late February. The infection was light and caused a moderate defoliation of the plants. Specimens have been deposited in the herbarium of the Plant Pathology Department, Cornell University, Ithaca, New York.

DIRECCIÓN GENERAL DE INVESTIGACIONES AGRONÓMICAS, MINISTERIO DE AGRICULTURA Y GANADERIA, SANTA TECLA, EL SALVADOR, C. A.

[1]Plant Pathology Department, Dirección General de Investigaciones Agronómicas, Santa Tecla.
[2]Identification of the species kindly made by Dr. Charles Chupp.

BOOK REVIEW

"MONOGRAPH OF MONOCHAETIA AND PESTALOTIA," by Emil F. Guba, Harvard University Press, Cambridge, Mass. vii + 342 pages, 125 text figures. 1961. Price $10.00.

The genus Pestalotia has long been one of those classic catch-alls to which alleged new species have been added indiscriminately on the mere excuse of a new host or substrate or a new geographical area with little attention to comparative morphology or possible earlier descriptions. Accordingly there has been need of a critical and comprehensive monographic study of the genus and of related genera characterized by multiseptate, setulate conidia with intermediate colored cells and exterior hyaline cells. This need Dr. Guba has supplied, based on his studies of the group for nearly 40 years, during which time he has patiently reviewed past work, studied all available material from around the world and has presented here a comprehensive account of the complex involved.

Many of the species have been found associated with plant diseases, although none have proved of major importance. Others are present in soils or have been found capable of deterioration of wood, paper, textiles, and other plant and animal products, so that as a group they are of economic concern.

In a preliminary chapter the author discusses the nomenclature of Monochaetia and Pestalotia, noting that the former must be conserved. Thereafter he presents available data on cultural studies, economic importance with special attention to pathological tendencies, ascigerous forms, the setulae so important in classification, and finally gives a general outline of the comparative morphology of the fungi as an aid in the use of the keys used to separate the species. These keys are based entirely on conidial characters including size, number and coloration of the cells and number and varying characters of the setulae which crown the conidia. They emphasize the author's position that specific distinctions are to be based on fungus morphology and not on host, substrate, or geographic distribution.

In the systematic section Monochaetia is first considered, 41 species being recognized. Each species is described following a uniform pattern, synonyms are listed and pertinent literature cited. Hosts or substrate, geographic distribution and general comments helpful to an understanding of each species are included. Two hundred and twenty-one species of Pestalotia are treated in the same manner. The conidia of many species in both genera are illustrated. There follows a section on "indefinite species," including those inadequately described and for which study material was not available. Fifty-six species excluded from Monochaetia and Pestalotia are discussed in detail and their proper placing indicated.

A combined host and fungus index completes the volume. -- JOHN A. STEVENSON